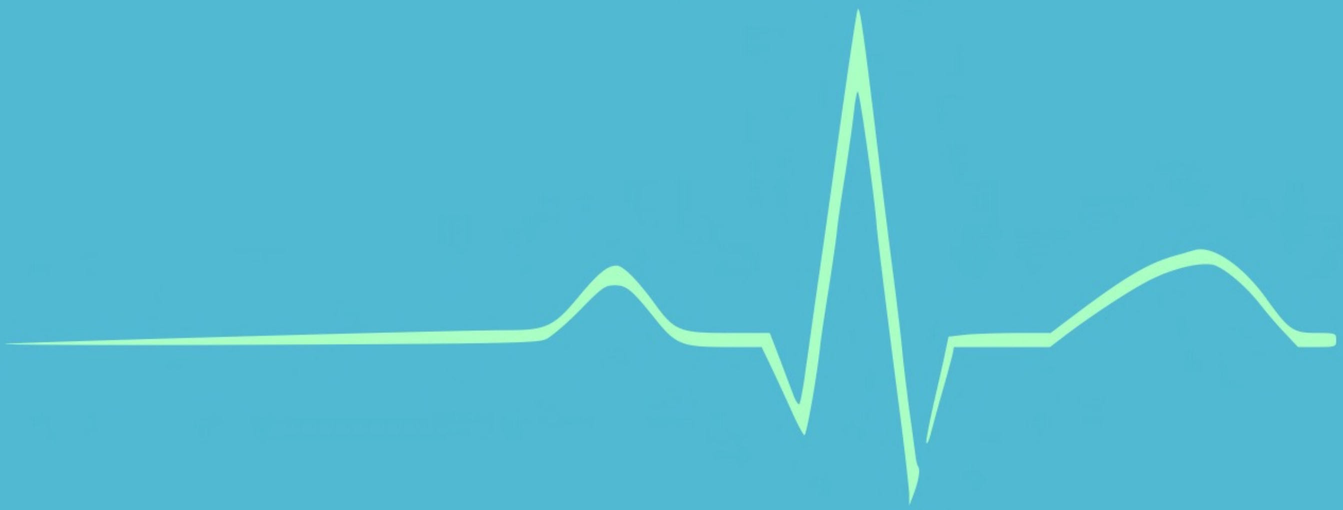
ECG / ABG & Miscellaneous

Based on Dr. Rakesh Nair's Marrow 4.0 Lecture



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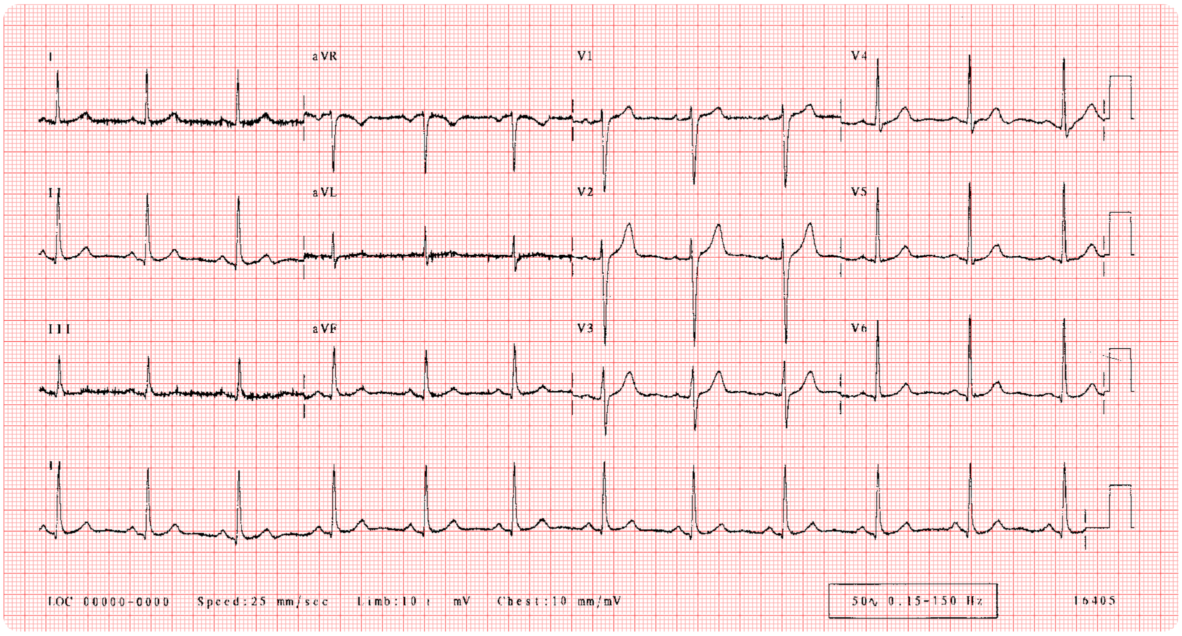
BASICS OF ECG

CATOGERIES OF ECG

1. Tachyarrhythmias
2. Brady arrhythmias
3. Localizing MI
4. Miscellaneous

ANALYSIS OF ECG



✓ Whenever the question is looking for an arrhythmia as the answer focus on RHYTHUM STRIP. (It's always the lead II)

✓ First look on STEP-1

QRS Complex
→ Narrow (normal) → Duration of QRS is <100 ms
→ width = 2.5-3 small boxes on ECG
→ Wide → Duration of QRS is >120ms
→ width = >3 small boxes on ECG

↓ stands for

VENTRICULAR DEPOLARISATION

↓ occurs in

NOTE: Every small boxes on ECG = 40ms = 0.04s

(A) SEPTAL DEPOLARISATION

→ Always proceeds from Lt ⟶ Rt
(∵ Lt bundle branch is shorter)
(∴ Rt leads will be positive)

→ V_1 (Rt leads) : small positive R-wave

V_5-V_6 (Lt leads) : small negative Q-wave

→ Small process ∴ deflection is also minimal

(B) MYOCARDIAL DEPOLARISATION

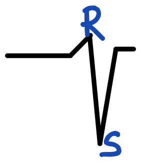
→ Occurs as SANode
↓
Atrium
↓
AV node
↓
Bundle of His
↓ ↓
Rt. Bundle Branch | Lt. Bundle branch
↓ ↓
Lt. Ant. fasicle | Lt. Post. Fasicle

→ Both these synchronously cause Ventricular depolarisation.
FROM ENDOCARDIUM ⟶ MYOCARDIUM

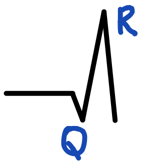
ENDOCARDIUM ↓ Purkinji fibers situated (∴ conduction is fast) ↓ ∴ Narrow QRS complex

MYOCARDIUM ↓ Bulckiest part ↓ ∴ Bigger deflection

→ Direction is from Rt ⟶ Lt (∵ Lt sided leads will be positive)
∴ in V_5-V_6 : Prominent R wave
in V_1 : Prominent S wave



IN V_1:
- small R wave
- prominent S wave



IN V_5, V_6:
- small Q wave
- prominent R wave
- no S wave

(C) POSTEROBASAL LV & POSTEROBASAL SEPTUM

→ 3rd parts to get depolarised
→ Terminal part of Ventricular depolarisation: APEX to BASE

WIDE QRS COMPLEX

- That means conduction not occured via Purkinji System

CONDITIONS IN WHICH WIDE QRS COMPLEX PRESENT

(a) Ventricular Pre excitation
- ventricular premature complex (or) Ventricular tachycardia
- Ventricle is actually the source of Rhythm or the focus → WIDE QRS

(b) Aberrant Pathway
- Accessory pathway present → activate ventricle : WPW Syndrome

(C) Bundle Branch block
- Either Left or Right

STEP-2 : Check it's Sinus Rhythum or what?

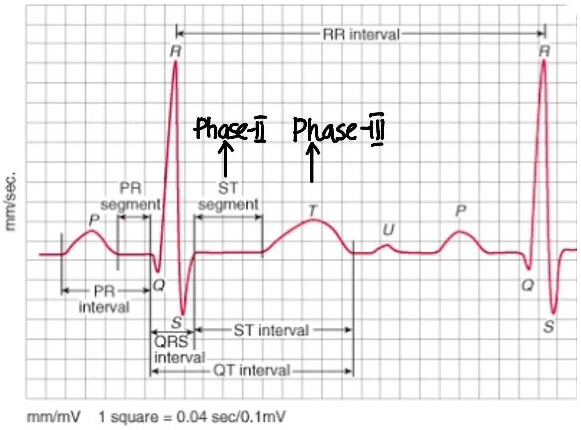
✓ Now check : QRS Complexes are preceeded by P wave
↓
i.e Ventricular activation occured from above

✓ Next Look into : PR interval ⟶ 3-5 small boxes = 120 - 200 ms
→ Beginnig of P wave to beginning of QRS
→ P wave + PR segment

P wave ↓ Denotes the activation of atria

PR segment ↓ Time taken to impulse to move from Atria to AV node, inside AV node & Upper part of Bundle of his

SA Node
↓
Atrium — P wave
↓
AV node — PR segment
↓
Bundle of His



→ Short PR interval
: <120 ms = <3 small boxes

→ Prolonged PR interval
: >200 ms = >5 small boxes

Impulse can be originated from

SINUS : Normal Morphology P wave
OR
ATRIUM : Abnormal Morphology P wave
OR
AV NODE : No P wave
OR
VENTRICLE : QRS complex is wide

AV Nodal Delay :
- Normal phenomenomenon
- When the impulse travels across AV node it take sometime
- i.e the reason the PR interval is b/w 3-5 small boxes

PROLONG PR INTERVAL ⟶ AV BLOCK

SHORTENED PR INTERVAL ⟶ WPW SYNDROME
(Aberrant pathway connect Atria to ventricle bypassing the AV node)

STEP-3 : Check it's Regular or Irregular

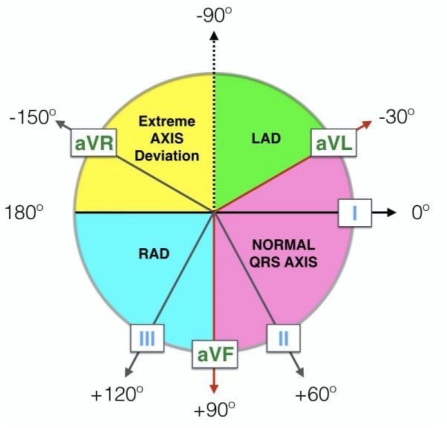
✓ Look for : R-R interval is regular or not

STEP-4 : Calculation of Rate

$$\text{Rate} = \frac{1500}{\text{no. of small boxes b/w two R waves}}$$

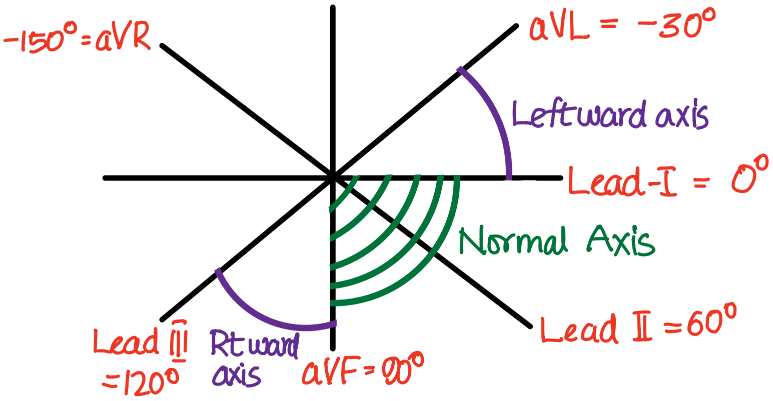
∴ The first ECG is of Normal Sinus Rhythum, Regular with narrow QRS Complex, Rate of 70/minutes.

STEP-5 : Calculation of Axis of ECG



For calculation of axis look into
- Limb leads
- Augmented Limb leads

DONT LOOK INTO CHEST LEADS



- Anything more than -30° = Left Axis Deviation (LAD)
- Anything more than 120° = Right Axis Deviation (RAD)

- Normal axis = 0-90°
- Leftward axis = 0-(-30°)
- Rightward axis = 90-120°

✓ Calculate the voltage of Lead I, II, III, aVL, aVF. (R-wave deflection)

From the 1st ECG : 12 15 8 7 10 small boxes

2nd maximum Max Voltage

0° 60°

∴ Axis of ECG is b/w 0° & 60° (More likely b/w 30-60° as Lead II (60°) is max)

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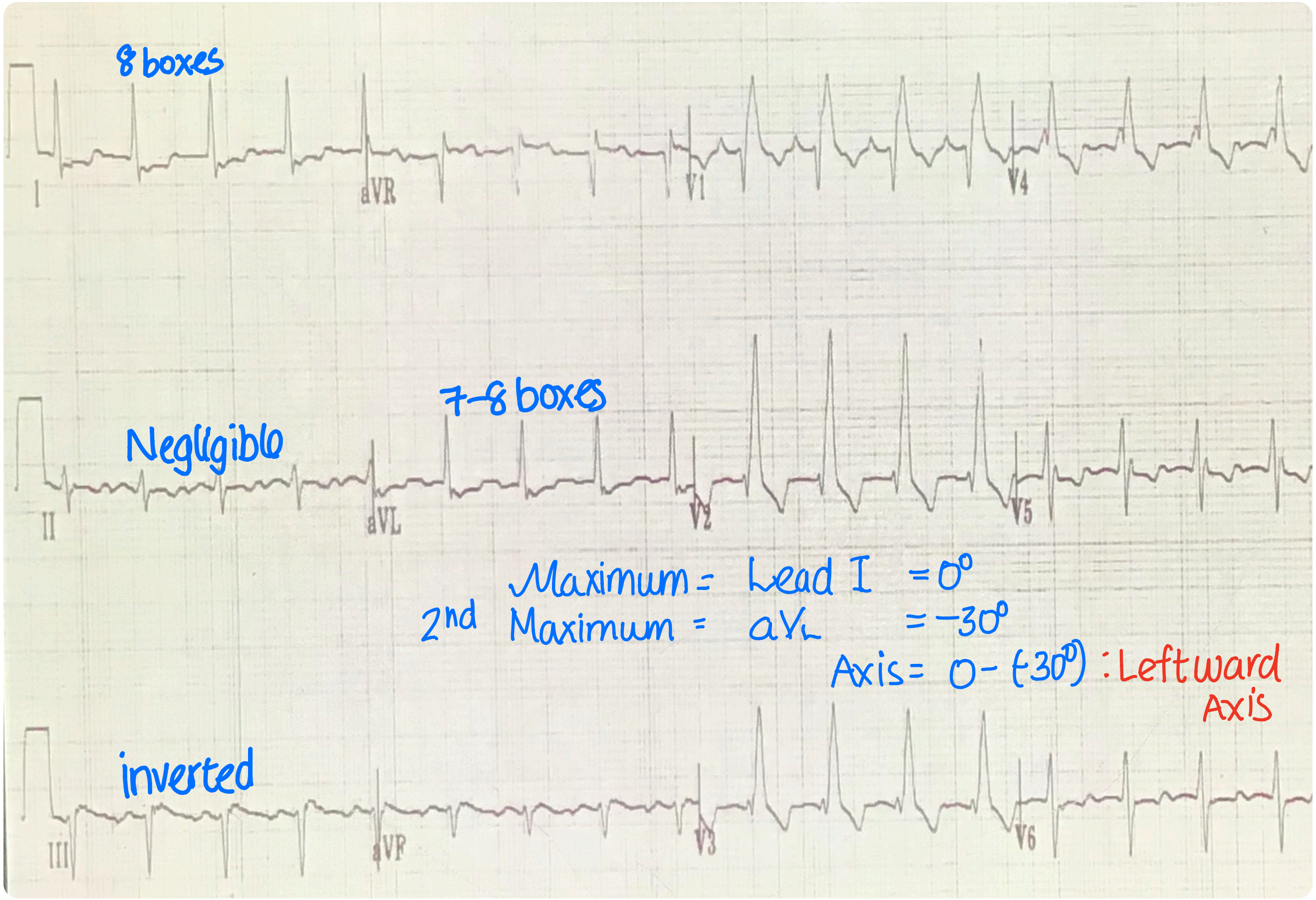
? Calculate the axis of following ECG



8 boxes

Negligible

7-8 boxes

inverted

Maximum = Lead I = 0°

2nd Maximum = aVL = $-30°$

Axis = $0 - (-30°)$: Leftward Axis

STEP-6 : QT Interval

- Some total of Ventricular activity

↓

Ventricular depolarisation (from endocardium to epicardium)

+

Ventricular Repolarisation (from epicardium to endo cardium)

- from beginning of QRS complex to end of T wave or U wave

- Normal QT interval = 0.40 – 0.42s = 10-11 small boxes
- Prolong QT interval = >12 small boxes

female is having prolong QT interval → 0.46 s ♂
0.48s ♀

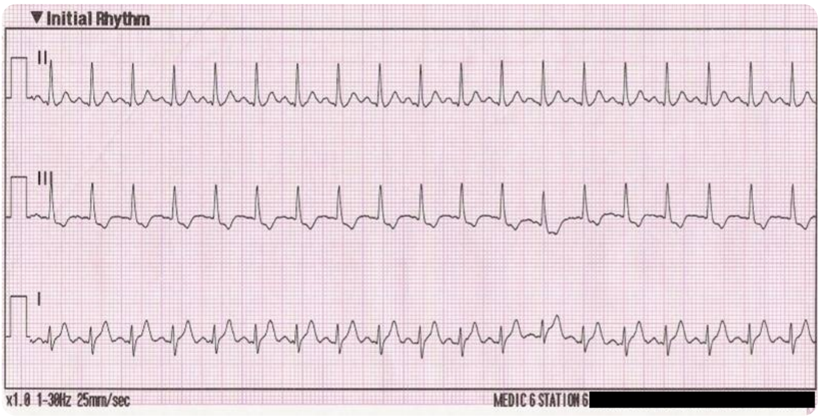
CORRECTED QT INTERVAL (QTc) $= \frac{QT}{\sqrt{RR}}$

In the First graph : QT = 10 small boxes = 400ms = 0.40s
: RR = 20 small boxes = 800ms = 0.80s

$QTc = \frac{0.40}{\sqrt{0.80}}$

QT Prolongation is due to

- Prolongation of Ventricular Repolarisation (Phase-III Cardiac A·P Prolongation)



- Concentrate on Rhythum strip

STEP-1 : NARROW QRS (supraventricular impulse)

STEP-2 : Every QRS preceeded by Pwave
: PR interval normal

STEP-3 : Regular

STEP-4 : 1500/10 =~150 : Tachycardia
Narrow QRS complex Tachycardia.

Sites of origin of Impulse can be

Above it's a Sinus Tachy. : Normal P wave ⟵ SINUS ⟶ Sinus Tachycardia
or
Abnormal P wave ⟵ ATRIA ⟶ Atrial Tachycardia
or
No Pwave ⟵ AV Node ⟶ Junctional Tachycardia

- MC Tachycardia
- MC Supra ventricular Tachycardia
- MC Narrow QRS Complex Tachycardia
- Rate : <180/minutes
  if it's >180/minutes sinus will not be able to control the impulse.

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NARROW QRS COMPLEX TACHYCARDIA

TACHYCARDIA : Rate of ECG >100/minutes

↓

QRS Complex → Narrow

QRS Complex → Wide → (i) VT
(ii) WPW Syndrome (Antidromic)
(iii) Bundle Branch block in SVT

NARROW QRS COMPLEX TACHYCARDIA

BASED ON ORIGIN

(i) SINUS TACHYCARDIA
(ii) FOCAL ATRIAL TACHY.
(iii) MULTIFOCAL ATRIAL TACHY. (MAT)
(iv) JUNCTIONAL TACHYCARDIA
(Non paroxysomal)
(v) RE-ENTRY TACHYCARDIA (RT)
- AV Nodal RT (AVNRT)
- Atrio Ventricular RT (AVRT)
- Sino atrial RT (SART)
- Inter atrial RT (IART)

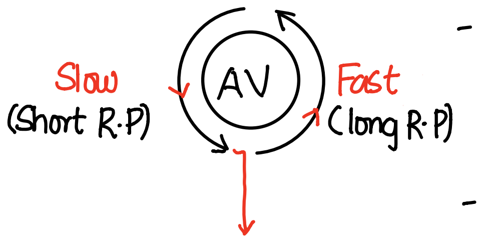
(vi) Atrial Flutter
(vii) Atrial Fibrillation

BASED ON MECHANISM

(i) RE-ENTRY

- Promble in impulse conduction, not in impulse initiation
- 1 single premature atrial impulse that is causing Re-entry

In AVNRT



- Re-entry circuit across AV Node.
- There are two pathways across AV node
  (i) Slow pathway : short Refractory period.
  (ii) Fast pathway : Long Refractory period

- Perfectly timed premature atrial complex (trigger) comes to upper part of AV Node

↓

impulse enter into short pathway (∵ short R·P)

↓

Reaches the upper part of Bundle of His

↓

By the time Fast pathway has recovered, ∴ one impulse conducted downwards & other going through fast pathway.

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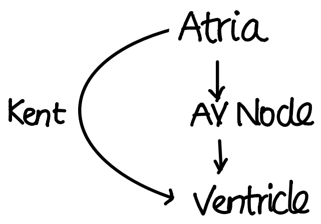
In AVRT



- Always associated with WPW Syndrome
- Perfectly timed premature atrial complex get produced when AV node has recovered & bundle of Kent is Refractory

↓

impulse will travel down to AV Node & activation of ventricle.

↓

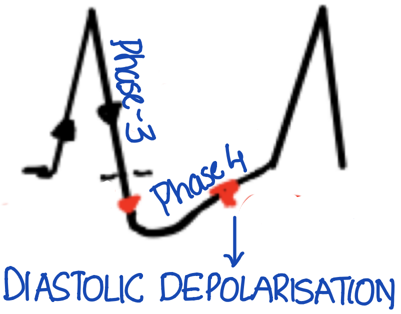
At this time Kent get recovered & some impulses traverse back to atrium

✓ In RT, it's the conduction that across AV node is predominantly the issue

So use VAGAL MANEUVERS
AV NODAL BLOCKING AGENT
Electrical DC Cardioversion is also recommended

✓ In structurally normal Heart, RE-ENTRY TACHYCARDIA is MC

(ii) ENHANCED AUTOMATICITY



In Pacemaker Cells

✓ Slope of Phase-4 : Rate of Automaticity

Enhanced automaticity or Enhanced Phase-4 depolarisation } TRIGGER ARRHYTHMIA

Eg : Atrial tachycardia (Focal & Multifocal)
Junctional tachycardia

(i) Problem of ↑ in firing Rate of Automatic cells. No RE-ENTRY circuit. Problem with INITIATION OF IMPULSE

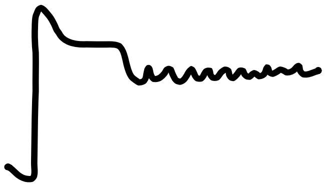
(ii) Vagal maneuvers
AV node Blockers } No use

(iii) Electrical cardioversion : No USE

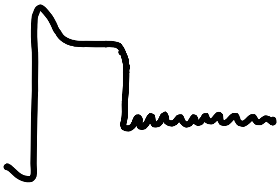
(iv) Most often occurs in structurally abnormal hearts

(iii) TRIGGERED ACTIVITY

- most often due to AFTER DEPOLARISATION



Early After depolarisation (EAD)



Delayed after Depolarisation (DAD)

EAD & DAD can triggers arrhythmia

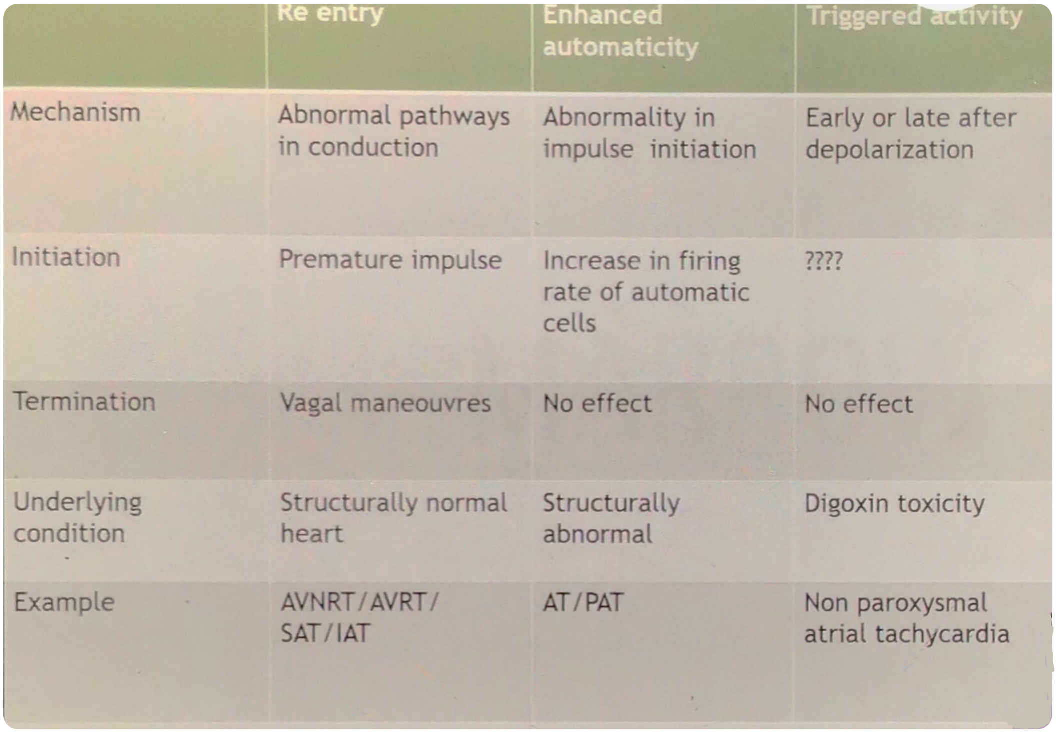
(i) Triggered activity/EAD : Predominant mechanism of arrhythmia

↓

DUE TO DIGOXIN TOXICITY

(i) Atrial tachycardia ̅c 2:1 block

(ii) Non peroxysomal Junctional tachy- with block.



	Re entry	Enhanced automaticity	Triggered activity
Mechanism	Abnormal pathways in conduction	Abnormality in impulse initiation	Early or late after depolarization
Initiation	Premature impulse	Increase in firing rate of automatic cells	????
Termination	Vagal maneouvres	No effect	No effect
Underlying condition	Structurally normal heart	Structurally abnormal	Digoxin toxicity
Example	AVNRT/AVRT/ SAT/IAT	AT/PAT	Non paroxysmal atrial tachycardia

SUPRA VENTRICULAR TACHYCARDIAS

Narrow QRS Complex ECG

Check

Regular RR Interval

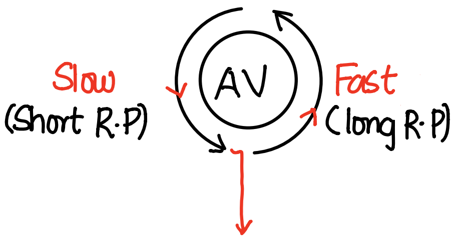
Eg: •AVNRT
- AVRT
- Junctional tachy.
- Sinus tachycardia
- Paroxysmal/Focal Atrial Tachy
- Atrial Flutter

Irregular RR interval

Eg: •Atrial fibrillation
- MAT
- Atrial Tachy ̅c block
- Atrial Flu ̅c block.

AVNRT - AV Nodal Re-entry Tachycardia

- MOST COMMON TYPE OF SVT DUE TO RE-ENTRY: AVNRT > AVRT > SART > IART
- MOST COMMON TYPE OF SVT : Sinus Tachycardia > Atr. Fibri > AVNRT
- Most often in Structurally normal heart
- Seen in Females > Males
- Heart Rate : 100-250/minute (MC : 180-250/minute)
- Perfectly timed premature atrial complex Comes to AV Node



↓

Impulse enter into short pathway (∵ short R·P)

↓

Reaches the upper part of Bundle of His

↓

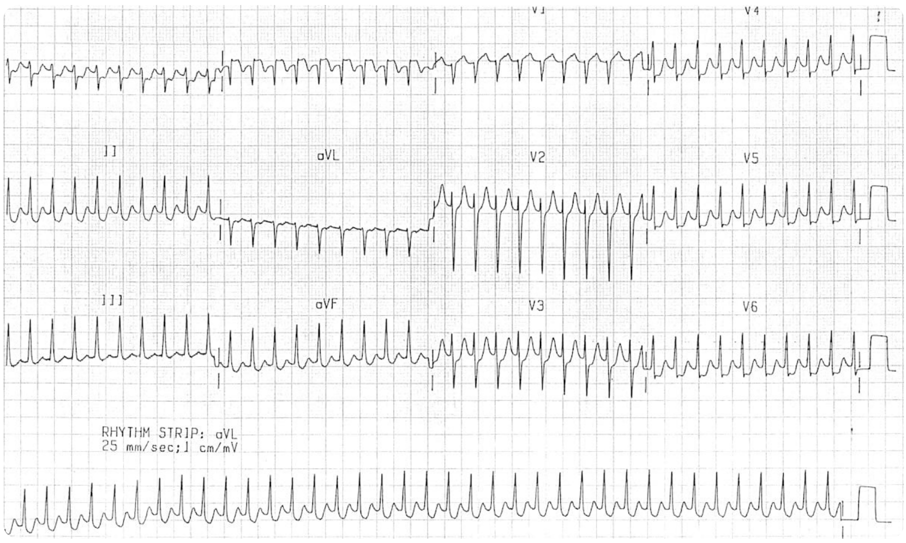
By the time Fast pathway has recovered, ∴ one impulse conducted downwards & other going through fast pathway.

↓ ANTEROGRADE ↓ RETROGRADE

- This leads to **SYNCHRONOUS ACTIVATION OF ATRIA & VENTRICLE**

↓

i.e $2/3^{rd}$ of Patients **P WAVE BURRIED INSIDE QRS COMPLEX**



- Narrow QRS Complex
- Rate : 1500/5 = 250-300/min
- RR interval = Regular

It's not

Sinus tachycardia ∵ no P wave

Atrial ∵ no P wave

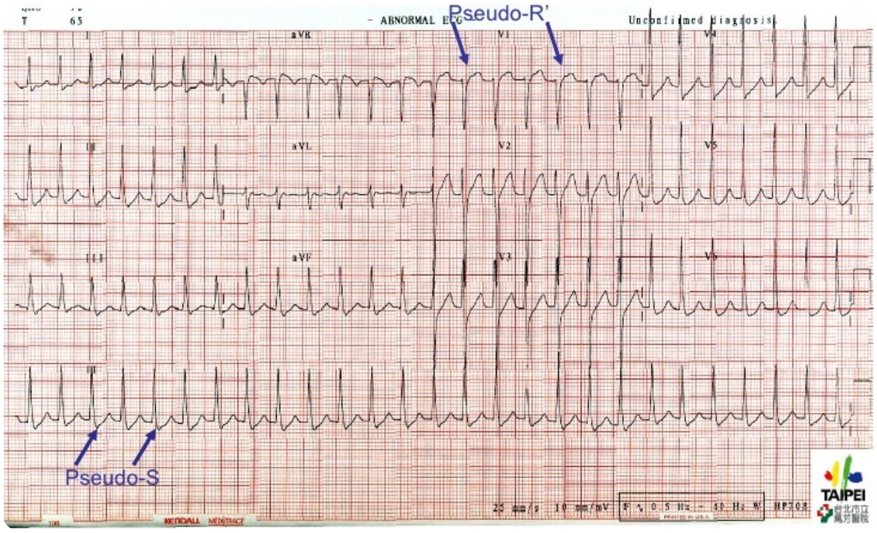
Flutter ∵ no flutter waves → Altered P waves

Junctional : it will be never >150/min usually <130/min

AVRT ✗ : No P wave fall outside.

It's AVNRT ✓ : Correct

S-F AVNRT



Narrow QRS Complex
Regular RR interval
No P waves
∴ AVNRT (or)
AVRT

✓ In AVNRT

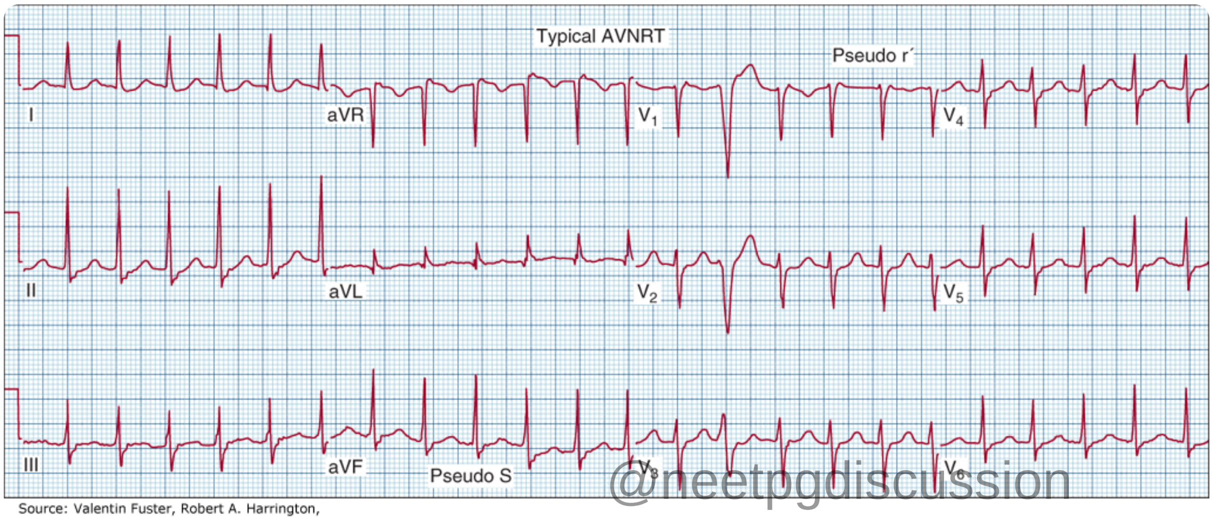
(i) 2/3rd cases : P waves buried inside QRS

(ii) 1/3rd cases : P waves just outside the QRS Complex
  (i) MC in the ascending limb of next T wave
  called as PSEUDO S-WAVE (MC)

  (ii) When it falls on the ascending limb of T wave of
  V1 called as PSEUDO R-WAVE

  (iii) When it falls before the QRS complex
  called as PSEUDO Q-WAVE (Least common)

NOTE : Whenever you see a small deflection
Calculate RP interval = <80ms (<2 small boxes)



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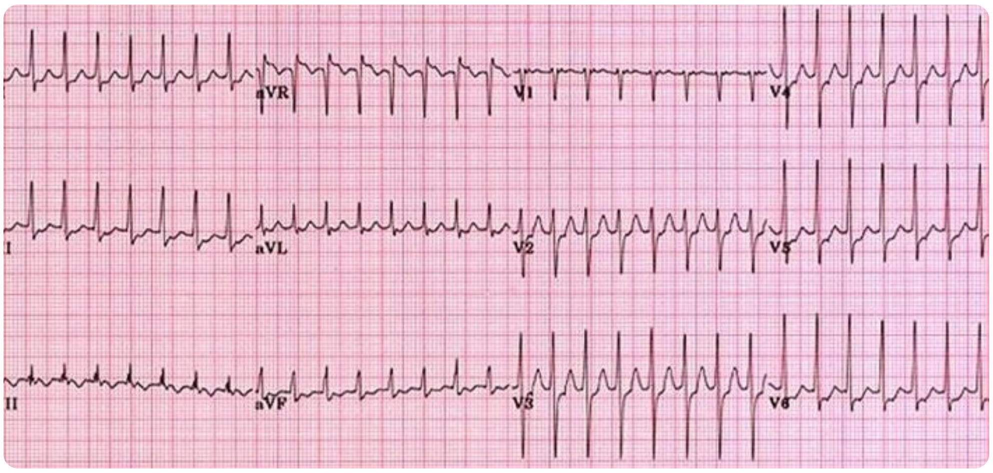


Narrow QRS Complex

Regular RR

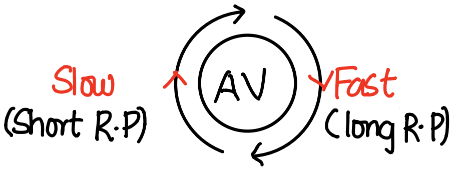
No pwave

AVNRT/AVRT

TYPICAL AVNRT (Slow-Fast AVNRT) in which
- (i) 2/3 No Pwave
- (ii) 1/3 Pseudo S, R, Q waves

ATYPICAL AVNRT (Fast-Slow AVNRT)

✓ Here Premature Ventricular complex
↓
Activation of atria by
- (i) RETROGRADE through SLOW Pathway
- (ii) ANTEROGRADE through FAST Pathway



✓ Here inverted Pwave falls before QRS complex

NOTE: Any Narrow QRS complex tachycardia with Regular RR interval Regardless of the Presence or absence of Reterograde Pwaves is AVNRT.

TREATMENT OF AVNRT, AVRT, AT

✓ VAGAL MANEUVERS:

(i) Carotid Sinus Massage

Identifying the carotid artery pulse
↓
Tracing carotid artery pulse to angle of mandible
↓
Gently apply pressure consistently with index & middle finger

(ii) Valsalva maneuvers

Pt to exhale against closed glottis & give a fist to abdomen & keep abdomen tensed.

(iii) DIVING REFLUX

Dip the head of of pt in cold water (vagal stimulation)

✓ DOC : Adenosine

- Blocks conduction across AV node
- $t_{1/2}$ = 10 sec
- Opens inward Rectifying K^+ Channels.

- Starting dose : 6mg i/v central vein or if peripheral: saline Chase i.e
  Through one port of canula: 6mg i/v adenosine
  Through other port : flux saline as soon as adenosine injected
  (do when patient is lying down)
  ↓
  Raise the upper limb
  ↓
  Turn the patient on prone position
- action within seconds
- if not responding try with 12mg i/v
  ↓ if not responded
  12mg i/v adenosine

- CONTRAINDICATIONS :
  (i) COPD or structural Lung disease as it cause bronchospasm

  (ii) Atrial fibrillation $\bar{c}$ WPW Syndrome
  blocking of AV node, ↑ impulse conduction through aberrant pathway

↓ NOT RESPONDING

Calcium Channel Blocker
(i) VERAPAMIL (ii) DILTIAZEM → Disadvantage :
- after giving as bolus dose, give continous maintainance infusion
→ Dose : 15-20mg

↓
- Preffered as it can be given as bolus dose
- start from 5mg, can give 3-4 times, max 20mg

↓ NOT RESPONDING

BETA BLOCKERS
METAPROLOL
- 2.5 - 5 mg i/v bolus
- can repeat 3-4 times

↓ NOT RESPONDING

CLASS Ia : PROCAINAMIDE
or
CLASS III : AMIODARONE

Patient with LV failure : β-Blocker & CCB C/I
↓
Adenosine not responding
↓
DIGOXIN 0.25-0.5mg
↓ NOT RESPONDING
CLASS III : AMIODARONE

NOTE : Any arrhythmia $\bar{c}$ Hemodynamic collapse
TOC : Electrical DC Cardioversion (Biphasic)

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AVRT : Atrio Ventricular Re-entrant Tachycardia

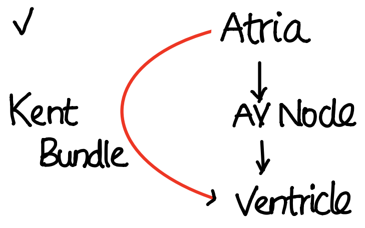
WPW SYNDROME

✓ 2 types

→ **Consealed** : No manifestation on ECG, no symptoms
First time patient present with AVRT
↓
Revert the AVRT to Normal
↓
Do Electrophysiological studies & find out WPW Syndrome

→ **Manifest** : Pt with WPW Manifestations on ECG
if the patient has symptoms + manifestation = WPW Syndrome
if the patient has only Manifestations = WPW Morphology

✓



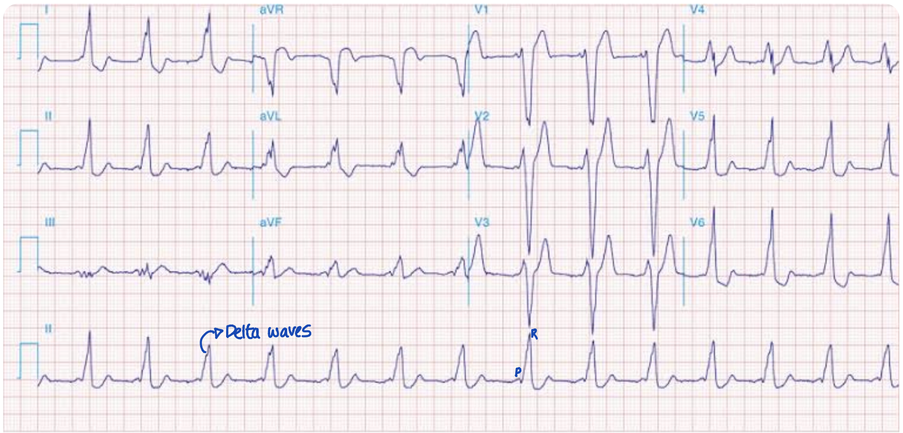
– Prerequisite for manifestation of WPW Syndrome
: Impulse transmission anterogradely (from Atria & Ventricle) across the aberrant tract / Bundle of Kent

In CONSEALED WPW : No impulse will travel through Aberrant / Bundle of Kent.
↓ patient
can manifest AVRT at any time

✓ ORTHOGRADE / TYPE A WPW SYNDROME :

- It goes anterogradely via the bypass tract & activate Ventricular myocardium
↓
∴ No AV nodal Delay ⇒ PR interval short
Ventricular myocardial activation is associated with **DELTA WAVE** via normal myocardial cell not via Purkinji cells

- Some impulse also travel through normal pathway with AV nodal delay
↓
∴ impulse reach the ventricle somewhat later. But this delay at the level of AV node can be compensated by activation of Purkinji System
↓
Catch up the impulse travels along bypass tract
↓
∴ QRS Complex more or less normal with terminal narrow deflection



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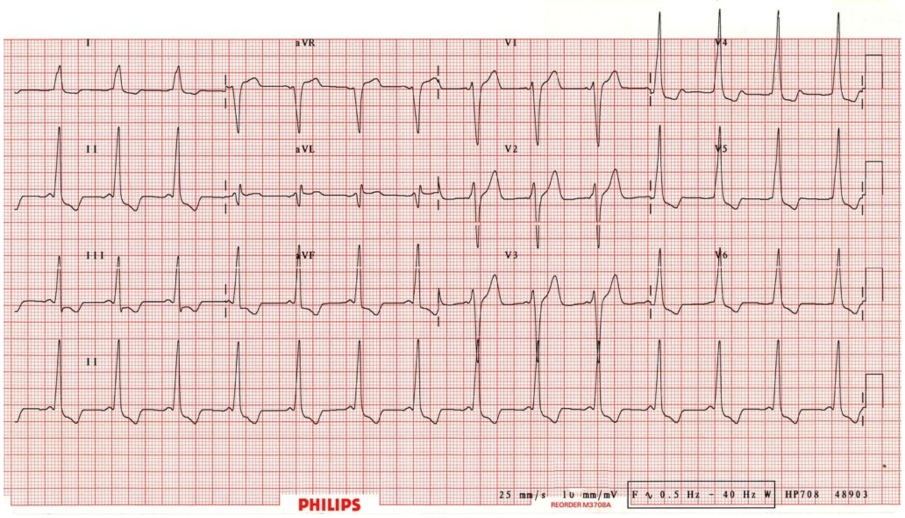
- Slightly wide QRS Complex
- Short PR interval

- Presence of Delta Waves
  ↓
  Upstroke of R wave showing deflection (due to initial Ventricular Depolarisation)
  or
  Terminal Narrow deflection of QRS Complex
  (due to catch up by Purkinji System)

EXAM POINTS ABOUT WPW SYNDROME

- Occurs at any age
- Male > Female

- Normal P wave
- Shortened PR interval
- Bizzarre delta waves
- Relatively normal narrow terminal QRS deflection
- Secondary ST & T wave changes



Near Normal QRS Complex

Short PR interval

Terminal Narrow deflection on QRS Complex

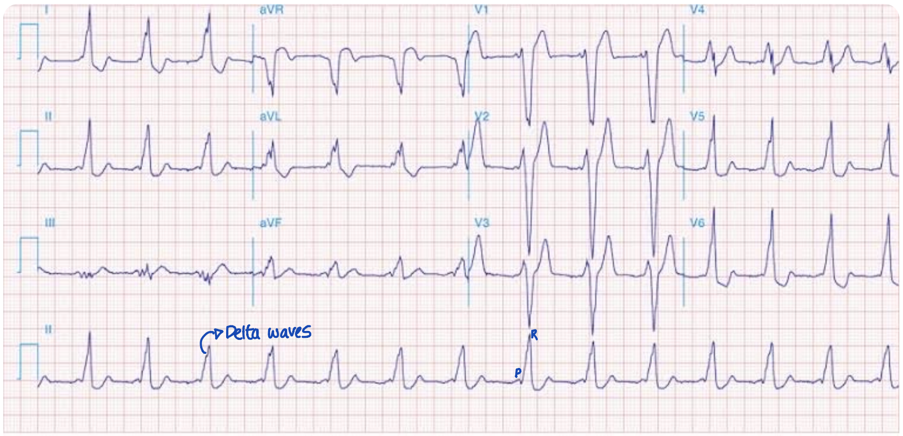
Secondary ST, T changes

WPW SYNDROME

LOCATION OF BYPASS TRACT

- Now find out the Bypass tract is (i) Lt Sided
  (ii) Rt sided
- Mostly it's left sided free wall > Lt sided Posteroseptal area > Rt sided
- In Lead V_1 : TALL R WAVES (or) PRESENCE OF DELTA WAVES } LEFT SIDED PATHWAY / TYPE A WPW

– In Lead V_1 : NEGATIVE WAVES : RIGHT SIDED PATHWAY / TYPE B WPW



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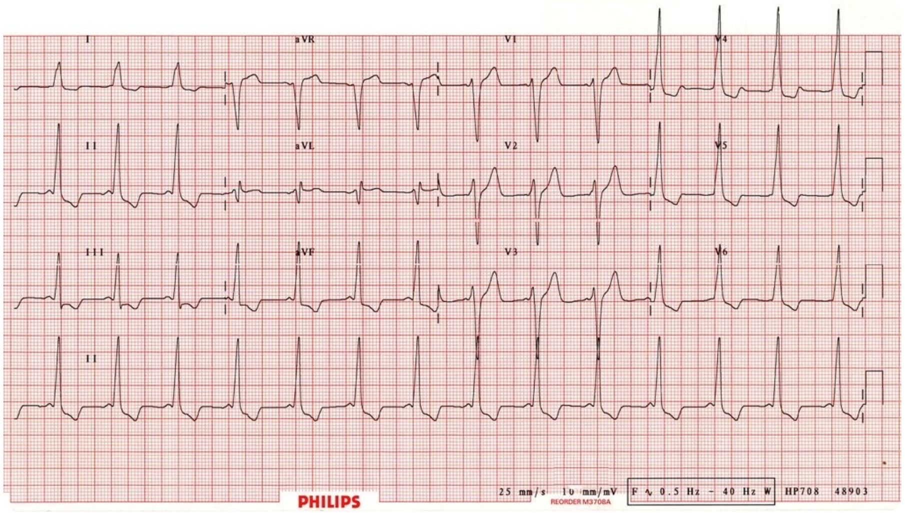
In V_1 :
NEGATIVE WAVES
↓
RIGHT SIDED / TYPE B WPW



In V_1 :
NEGATIVE WAVES
↓
RIGHT SIDED / TYPE B WPW

LOCATION OF LEFT SIDED WPW SYNDROME

– either on free wall or Posterobasal area

NEGATIVE or ISOELECTRIC : I, aVL, V_5 or V_6 ⇒ FREEWALL
NEGATIVE or ISOELECTRIC : II, III, aVF ⇒ POSTEROBASAL

✓ Size of DELTA WAVES depend upon amount of myocardium activated.

– Left sided tract has to travel longer distance
↓
Activates less of Myocardium
↓
∴ small Delta wave

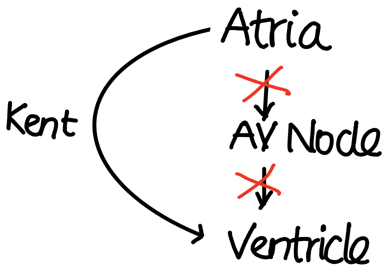
– Rt sided tract has to travel lesser distance
↓
Activates more of myocardium
↓
∴ Prominent DELTA WAVES

– Left sided path activates Lt side First
↓
∴ A2 occurs early. Then P2 (normal)
↓
A2-P2 interval widened = WIDE SPILT OF S2

– Rt sided path activates Rt side first
↓
∴ P2 occurs early. A2 occurs normal
↓
A2-P2 interval shortened = REVERSE SPLIT OF S2

ARRHYTHMIAS IN WPW SYNDROME

✓ 2 arrhythmias
(i) AVRT
(ii) Atrial Fibrillation

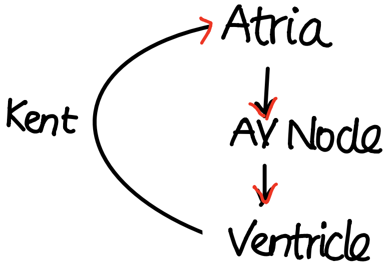


ANTIDROMIC AVRT

✓ ANTIDROMIC AVRT
- if the premature atrial complex passes through bypass tract & reach ventricle because the AV Nodal pathway is refractory

↓

We will get WIDE QRS Complex
(as it's not conducted through purkinji System)



ORTHODROMIC AVRT

✓ ORTHODROMIC AVRT
- if the impulses are all going via AV node & not going through bypass tract

↓

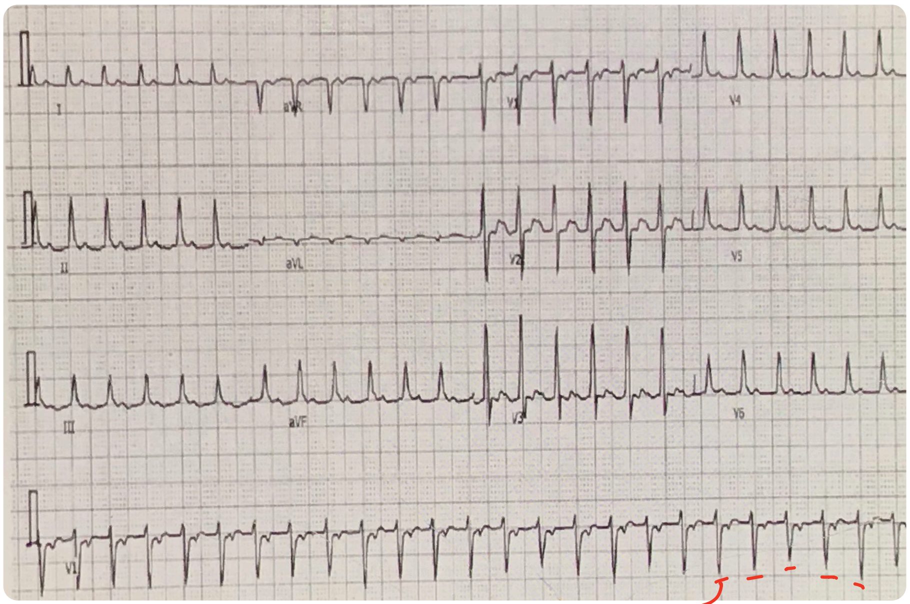
After reaching Ventricle, these impulse go reterogradely back to atria

↓

NARROW QRS Complex (No delta waves)
- Atria & Ventricle will not get activated synchronously

↓

P wave will fall outside the QRS complex



Electrical alternans

Narrow QRS Complex

Regular RR interval

No Normal Morphology P wave

↓

∴ AVNRT (or) AVRT

Pwave falling outside QRS
∴ AVNRT (or) AVRT

In an AVNRT
Pwave fall in ascending limb of T (most commonly) such that
RP interval is less than <80ms

In an AVRT
Seperately seen Pwave
RP interval is more than >80ms

Here RP interval >80ms ∴ AVRT

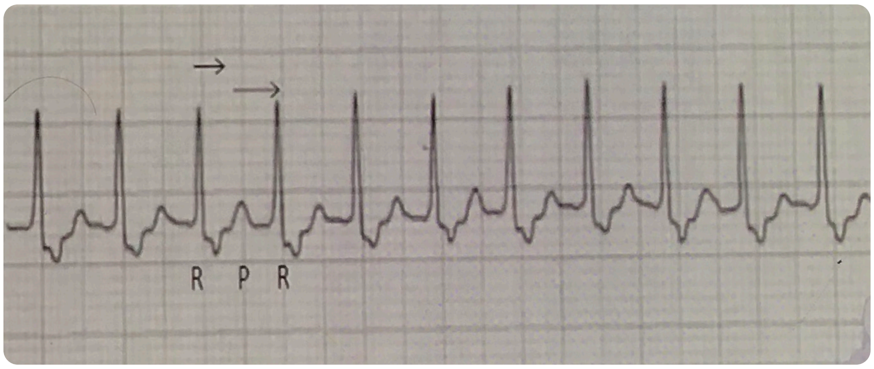
NOTE :

- If RP is short : AVNRT v/s AVRT

RP interval : <80ms ↓ AVNRT ; >80ms ↓ AVRT

- In AVRT
  RP interval - Short
  PR interval - Long

In Atrial Tachycardia
RP interval : Long
PR interval : Short



Narrow QRS Complex
Regular RR interval
Seperate P wave outside QRS

- PR > RP
  ↓
  ∴ it's not Atrial tachycardia
- RP interval > 80ms
  ↓
  ∴ it's AVRT

MANAGEMENT : AVRT

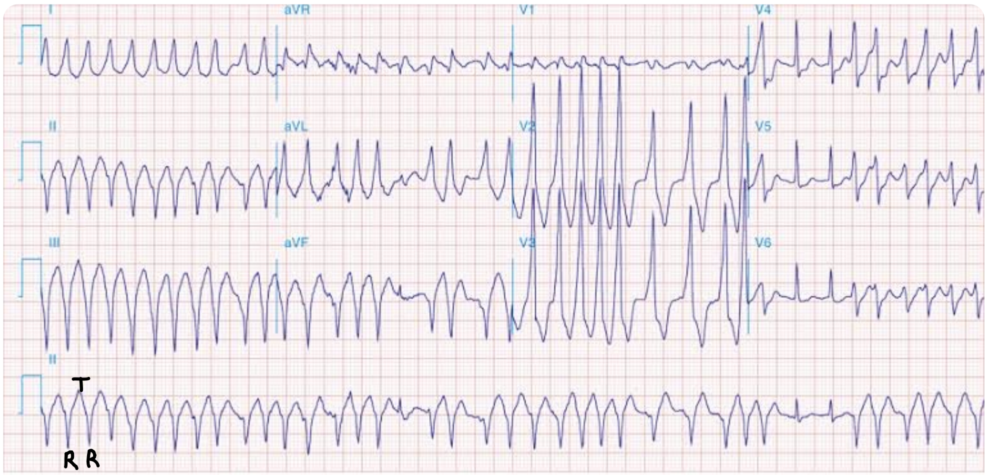
- acute management is same as that of AVNRT
- AVNRT occurs in structurally normal heart, but AVRT Require the patient take him to Electrophysiology & rule out WPW Syndrome

- In orthodromic AVRT : Adenosine
- Antidromic AVRT : Procainamide / Ibutilide (Ia or III)
- Radiofrequency ablation : Definitive treatment
- Recurrent Antidromic AVRT : FLECAINIDE

- WPW Syndrome with Atrial Fibrillation
  TOC : Electrical Cardioversion
  DOC : Procainamide (or) Ibutilide
  C/I : Never give drugs which blocks AV node (No digoxin, BB, CCBs)
  ↓
  Because it may precipitate V. Fibrillation

NOTE :

- Pt with concealed WPW syndrome present with ORTHODROMIC AVRT
- Pt with manifest WPW syndrome present with ANTIDROMIC AVRT



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Narrow QRS Complex
RR intervals are irregular
No morphological P wave
↓
Dx : Atrial Fibrillation in WPW Syndrome.

AVNRT	AVRT
- More common	- Less common
- Structurally normal heart	- Structurally abnormal heart
- No P wave seen	- P wave seen outside QRS
- if P wave seen RP <80ms	- P wave seen RP >80ms
- Blocks may seen	- No block in AVRT
- Electrical alterness is not seen	- Electrical alterness is seen

ATRIAL TACHYCARDIA

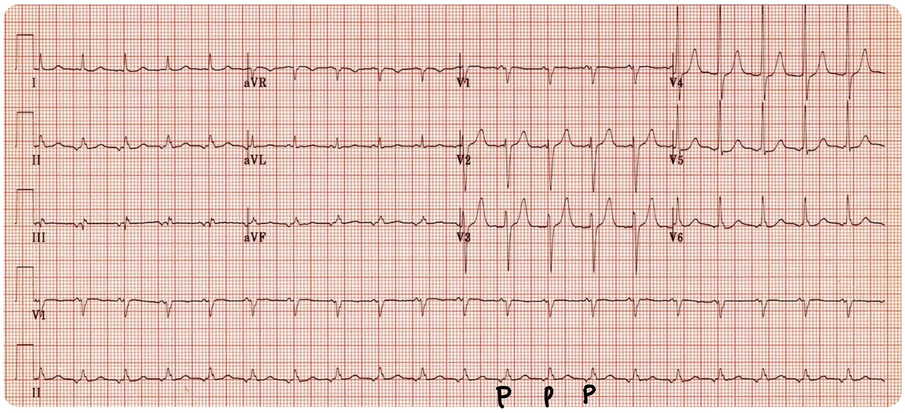
Narrow QRS Complex
↓
Regular RR Interval
↓
P wave definite normal morphology → SINUS TACHYCARDIA
No P wave → AVRT , AVNRT, JUNCTIONAL Tachy
Abnormal P wave → Atrial Tachy cardia

JUNCTIONAL Tachy ↓ Rate is much less

✓

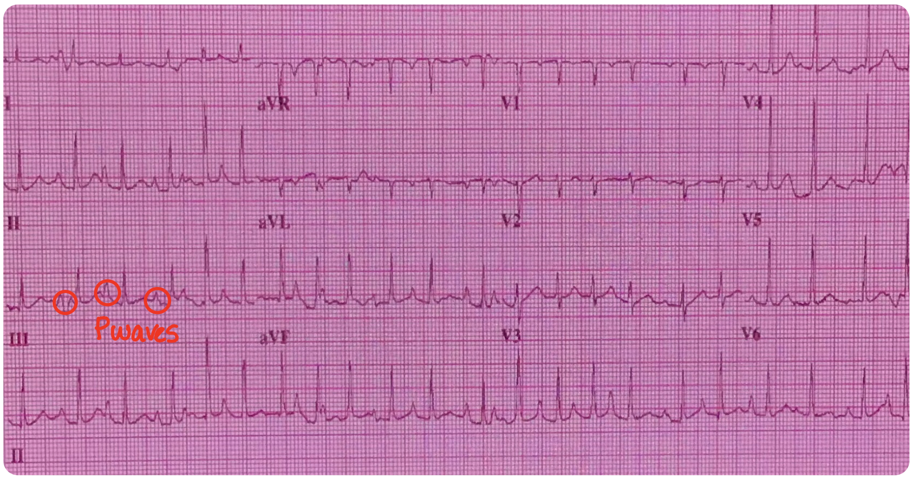
→ Focal / Paroxysmal Atrial Tachycardia (PAT) : Regular RR interval
EXCEPTION:
if there is block: Irregular RR interval

→ Multifocal Atrial Tachycardia (MAT) : Irregular RR interval



- Narrow QRS Complex
- Rate = $1500/11$ = 130-140 (generally low)
- Regular RR interval
- Small inverted Pwave before the QRS complex (abnormal Pwave)
- It's not Junctional: no Pwave RP > PR ∴ not AVNRT/AVRT

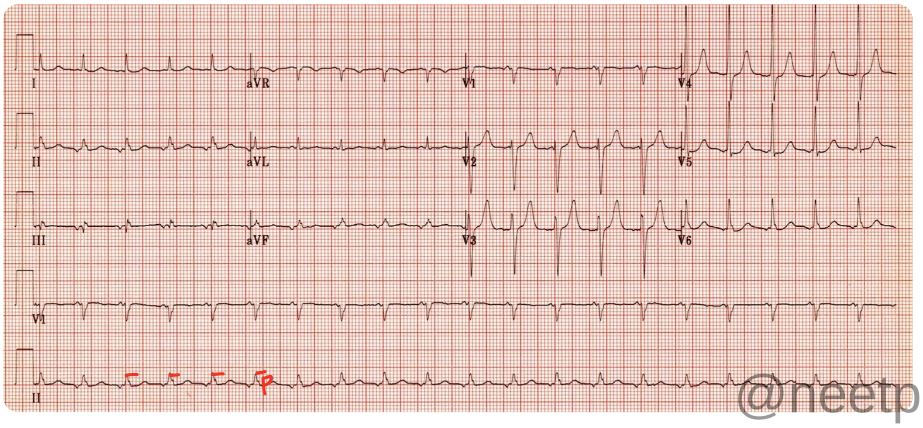
∴ It's focal Atrial Tachycardia.



- Narrow QRS Complex
- Irregular QRS complex
- 3 or more different P wave morphology

↓

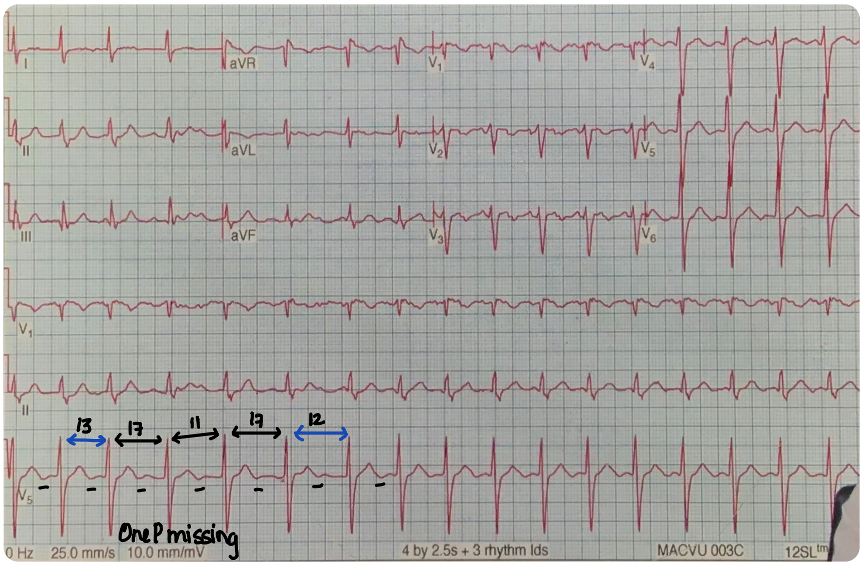
Dx: Multifocal Atrial tachycardia



- Narrow QRS complex
- Rate = $1500/12$ = 120-130
- Regular R-R interval
- Inverted Pwaves

↓

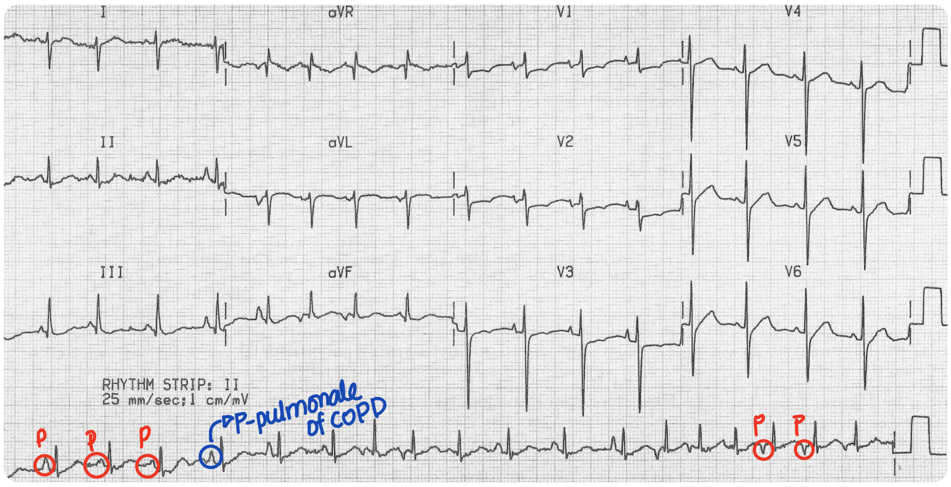
Dx: FAT



- Narrow QRS Complex
- Irregular RR interval
- P waves with same morphology some P waves are not conducted

↓

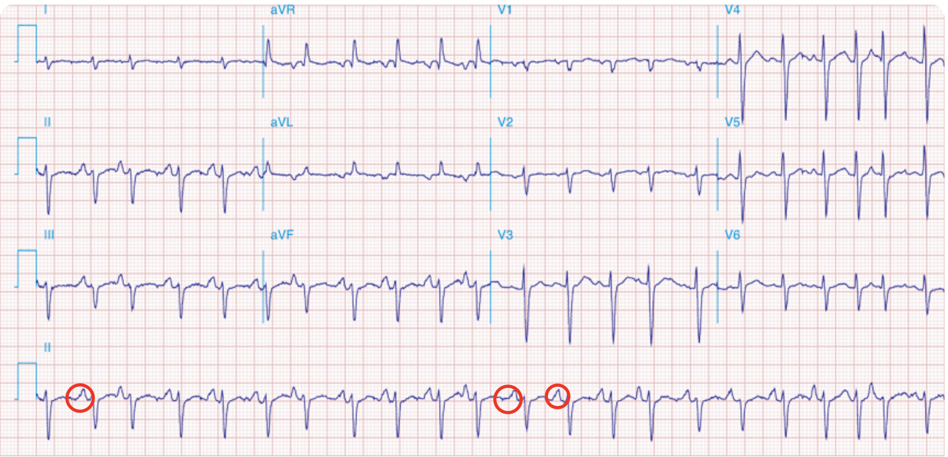
Atrial Tachycardia with block



- Narrow QRS complex
-
- 3 or more different types of P waves

Dx : MAT with additional features of COPD.

P-Pulmonale P wave : Tall P waves



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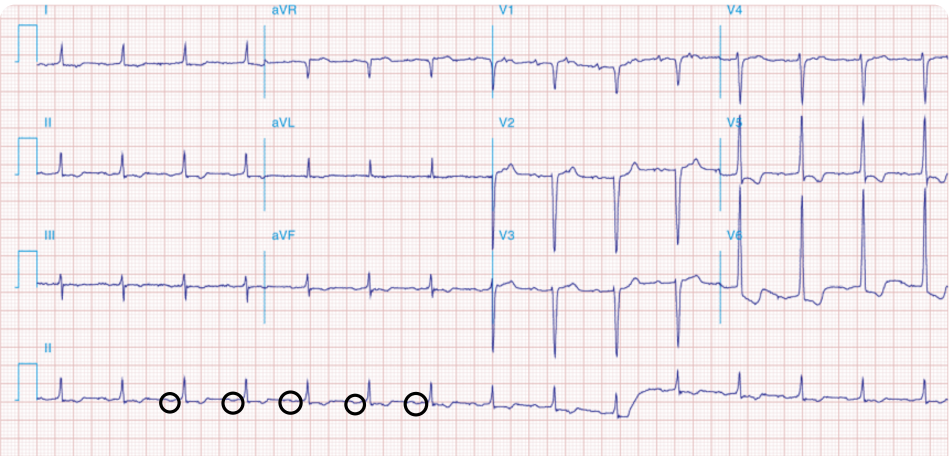
- Inverted QRS complexes
- Irregular RR interval
- 3or more different P wave morphology

↓

Dx : MAT



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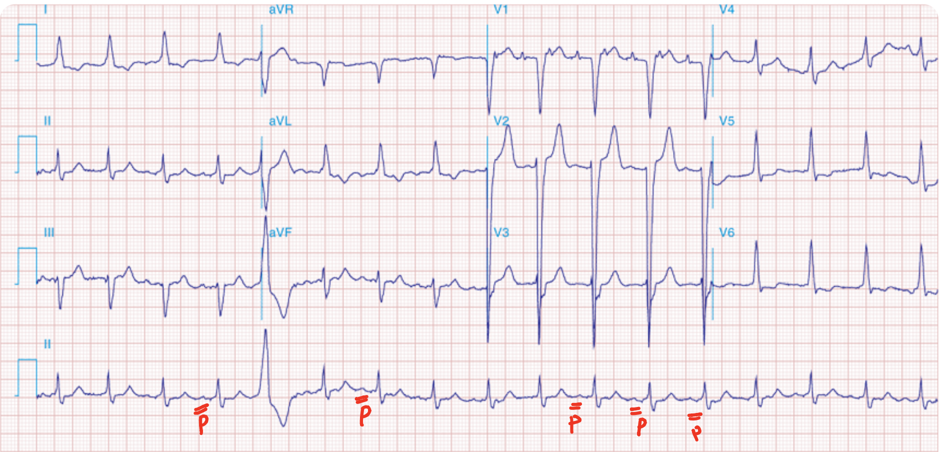
- Narrow QRS COMPLEX
- IRREGULAR R-R INTERVAL
- SAME MORPHOLOGY P WAVE
- SOME P WAVES ARE NOT CONDUCTED (2 paves only 1 conducted)

Dx: ATRIAL TACHYCARDIA WITH 2:1 BLOCK



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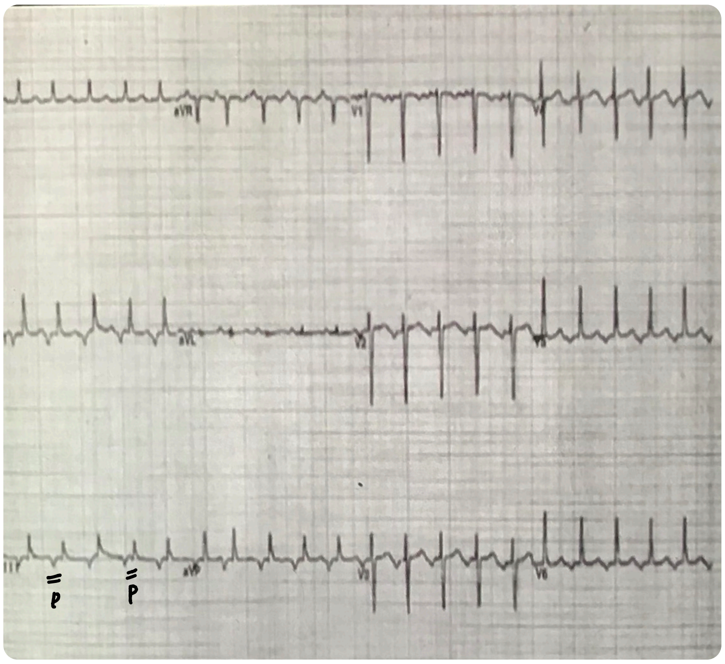
- NARROW QRS COMPLEX
- REGULAR RR INTERVAL
  Rate = 1500/15 = 100
- ALMOST SAME MORPHOLOGY P WAVE

↓

ATRIAL TACHYCARIA c̄ NO BLOCK



- INVERTED P WAVES
- NARROW QRS COMPLEX
- REGULAR R-R INTERVAL

↓

DIAGNOSIS : ATRIAL TACHYCARDIA

- Mechanism behind Atrial Tachycardia : ENHANCED AUTOMATICITY

↓

∴ AV NODAL BLOCKING AGENTS
VAGAL MANEUVERS
DC CARDIOVERSION
} will not Respond

- Here some kind of structural abnormality are present

MOST OFTEN OCCURS IN : MI
OBSTRUCTIVE LUNG DISEASE : (MC : MAT)

↓

When nebulising patient of COPD with HR >120/minutes leads to arrhythmia, Atrial tachycardia (MC : MAT)

ATRIAL TACHYCARDIA with 2:1 AV BLOCK ← DIGOXIN

↓

MECHANISM : TRIGGERED ACTIVITY

- Abnormal & discrete P waves
- Pathway is independent of AV CONDUCTION
- RATE is <200

- DEFINITIVE TREATMENT : Catheter Ablation

ECG FINDINGS & TREATMENT OF PAT

1. Regular Narrow QRS Complex tachycardia
2. P WAVES : UNIFORM IN CONFIGURATION BUT DIFFERENT IN MORPHOLOGY
3. ISOELECTRIC BASELINE BETWEEN 2P WAVES
4. PR INTERVAL : Prolonged
   VARYING Degrees of AV BLOCK Present

NOTE :

IRREGULAR RR → MAT
IRREGULAR RR → PAT $\bar{c}$ BLOCK

- ADENOSINE
  - Will not respond to Atrial tachycardia. But still we use because Rate will come down
  - But generally work in AVNRT, AVRT but not in AT.

- TREAT UNDERLYING PROBLEM (Structural Heart disease, COPD, Digoxin etc)
- REVERT THIS WITH CLASS I_A/I_C/III : PROCAINIDE/FLECAINIDE/IBUTILIDE/AMIODARONE
  DOC : IBUTILIDE 1mg i/v for 10 minutes
  with H.F : AMIODARONE

ECG FINDINGS & TREATMENT OF MAT

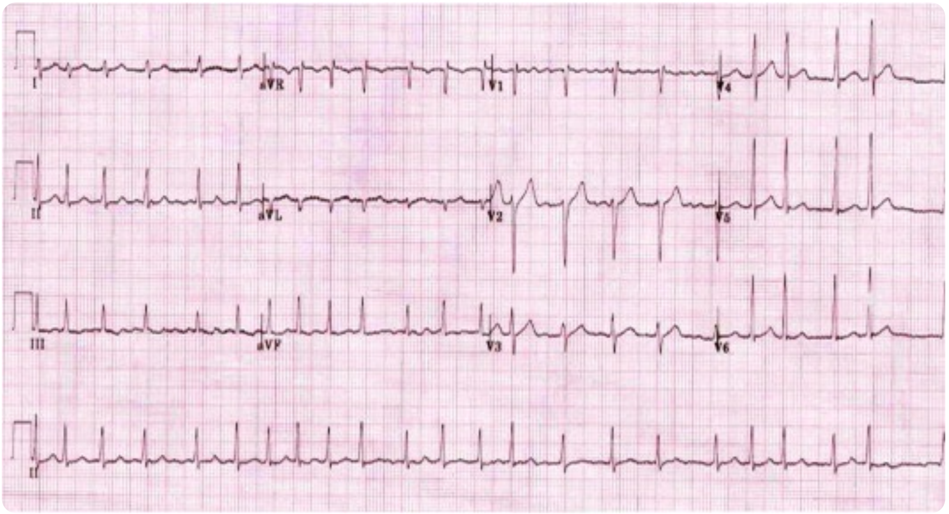
1. 3 or more different morphology P waves
2. ISOELECTRIC BASELINE
3. COPD/Theophylline

- TREAT UNDERLYING PROBLEM
- NO ROLE of ELECTRICAL CARDIOVERSION
- CALCIUM CHANNEL BLOCKERS/BETABLOCKERS USED

MAT V/S AF

	MAT	AF
*	doesnot require ANTICOAGULANT	Require ANTICOAGULANT
* 3or more different P wave morphology	✓	✗

ATRIAL FIBRILLATION



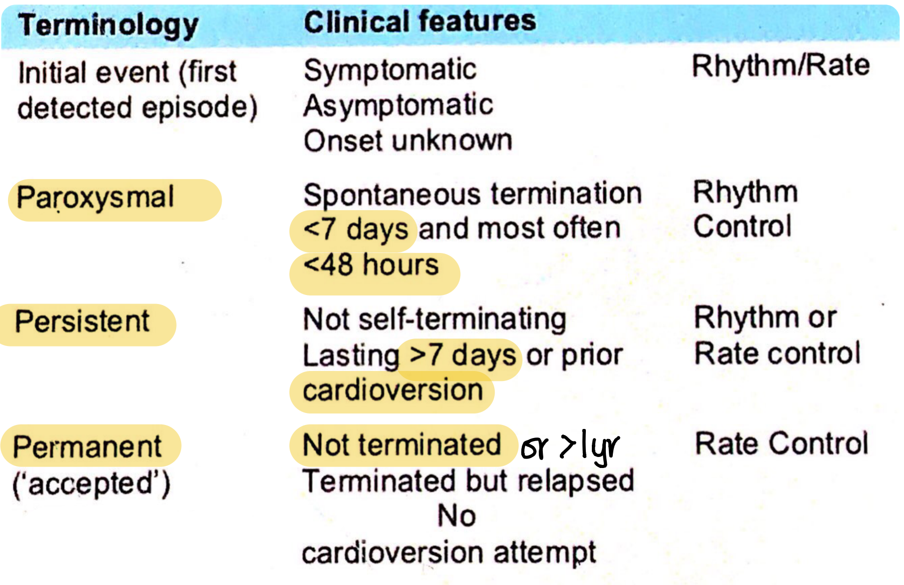
- NARROW QRS COMPLEX
- IRREGULAR RR INTERVALS
- NOT EVEN A SINGLE MORPHOLOGICALLY IDENTIFIABLE NORMAL P WAVE
- FIBRILLATORY WAVES

ATRIAL FIBRILLATION

- 3RD MC SVT
- MC RE-ENTRY ARRHYTHMIA

- HALLMARK : IRREGULARLY IRREGULAR Ventricular Rhythum
  Ventricular Rate : 400-450/min
  Atrial Rate : 100-160/min

CLASSIFICATION

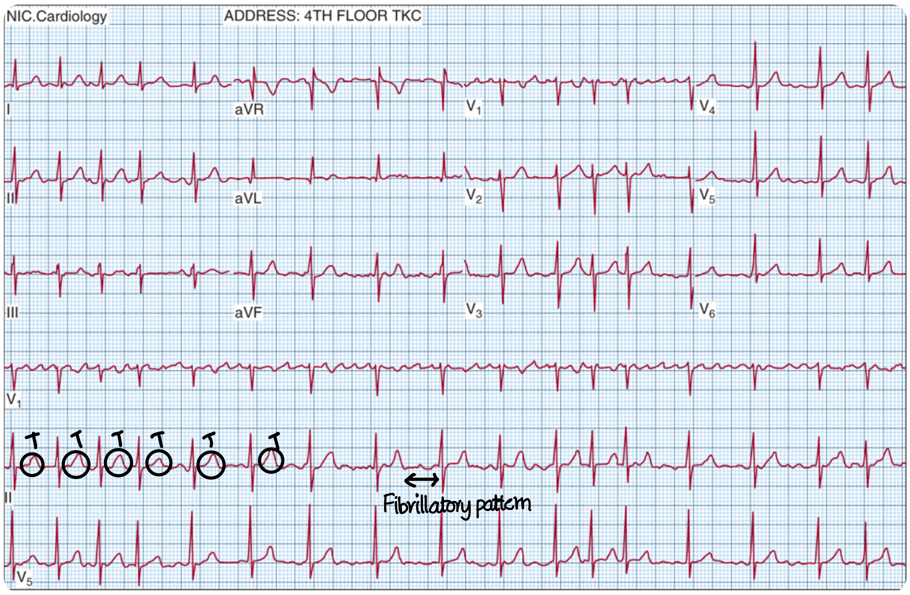


Terminology	Clinical features	
Initial event (first detected episode)	Symptomatic Asymptomatic Onset unknown	Rhythm/Rate
Paroxysmal	Spontaneous termination <7 days and most often <48 hours	Rhythm Control
Persistent	Not self-terminating Lasting >7 days or prior cardioversion	Rhythm or Rate control
Permanent ('accepted')	Not terminated or >1yr Terminated but relapsed No cardioversion attempt	Rate Control

LONE AF :
- AF which occurs <60yrs
- No structural heart disease
  No hemodynamic instability
  No co-morbidities

CAUSES OF ATRIAL FIBRILLATION

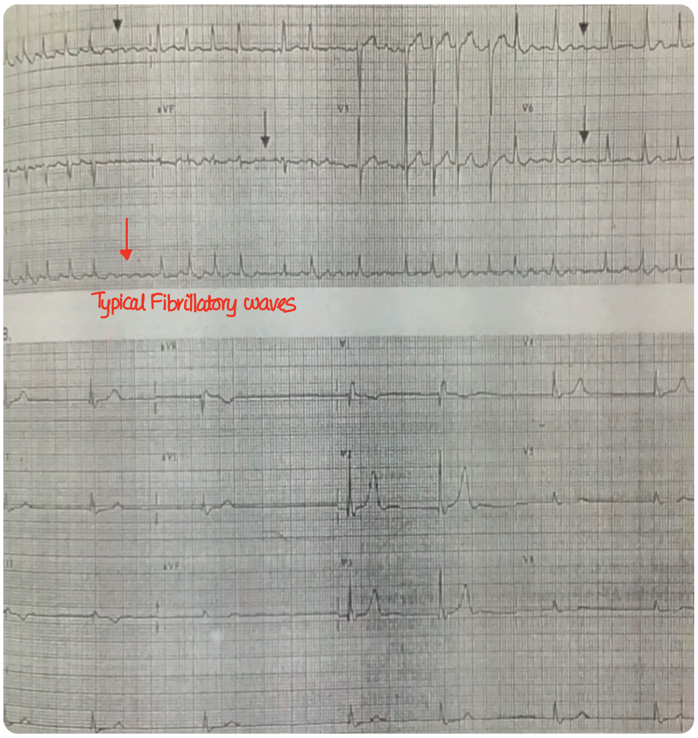
- MC Valvular heart disease causing AF: MR > MS
  - BOTH MS & MR associated with AF
    - MS ↓ MORE STRONGLY ASSOCIATED WITH AF
  - MR incidence is > MS
    - ∴ MC Valvular disease causing AF MR
- MC ACUTE CONDITION WHICH LEAD TO AF: PNEUMONIA (LRTI)
  ALCOHOL INTAKE
  THYROTOXICOSIS
- MC CONGENITAL HEART DISEASE ASSOCIATED WITH AF: ASD (Atrial Septal Defect)
- MC ARRHYTHMIA SEEN AFTER POST OPERATIVE SURGERY: ATRIAL FLUTTER > FIBRI...



- NARROW QRS COMPLEX
- IRREGULAR RR INTERVAL
- NOT EVEN A SINGLE MORPHOLOGICALLY IDENTIFIABLE NORMAL P WAVE
- FIBRILLATORY WAVES

↓

ATRIAL FIBRILLATION



- NARROW QRS COMPLEX
- IRREGULAR RR INTERVAL
- NOT EVEN A SINGLE MORPHOLOGICALLY IDENTIFIABLE NORMAL P WAVE
- FIBRILLATORY WAVES

↓

ATRIAL FIBRILLATION

HR IN ATRIAL FIBRILLATION

6-second Rule:

HR = N X10

N = No. of QRS Complexes in
30 large boxes or 6 seconds

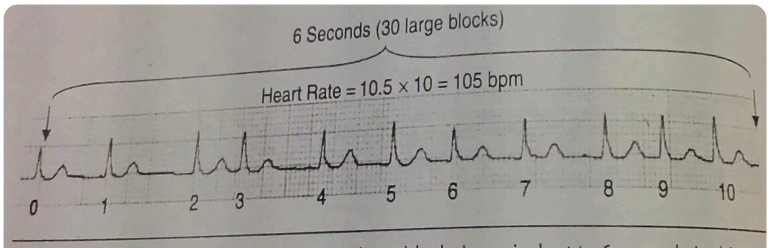


Figure 19.3: Six-Second Markers. Thirty large blocks is equivalent to 6 seconds. In this example, there are 10.5 QRS complexes within the 6-second time markers, thus the heart rate is 10.5 × 10 = 105 beats per minute.

ASHMANN PHENOMENON

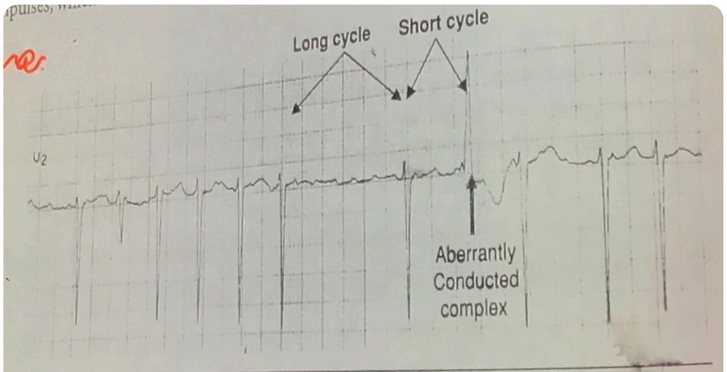


Figure 19.15: Ventricular Aberration. Arrow pointing up shows an aberrantly conducted supraventricular impulse, which is the second QRS complex after a long R-R interval. Aberrantly conducted complexes are wide usually with a right bundle branch block pattern. The wide QRS complex can be easily mistaken for a premature ventricular complex.

In AF after a LONG CYCLE when you are having a SHORT CYCLE, next IMPULSE IS FINDING VENTRICLE REFRACTORY (because it's not recovered from previous R.P)

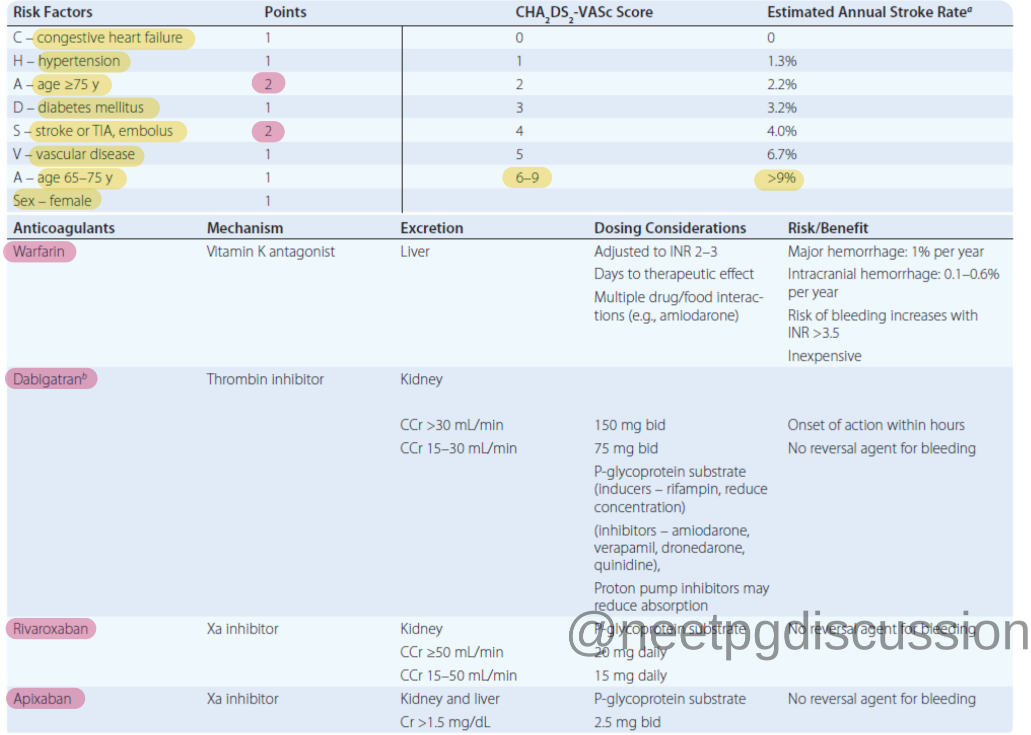
↓

NEXT IMPULSE WILL BE CONDUCTED DOWN THE VENTRICLE Called as ABERANCY

This Phenomenon is known as
ASHMANN PHENOMENON

CHADS$_2$-VAS SCORE

✓ ONE OF THE MOST COMMONEST CAUSE OF EMBOLIC STROKE IS AF
✓ CHADS$_2$ VAS SCORE Predict the RISK OF EMBOLIC STROKE after AF



Risk Factors	Points	CHA$_2$DS$_2$-VASc Score	Estimated Annual Stroke Rate[a]
C – congestive heart failure	1	0	0
H – hypertension	1	1	1.3%
A – age ≥75 y	2	2	2.2%
D – diabetes mellitus	1	3	3.2%
S – stroke or TIA, embolus	2	4	4.0%
V – vascular disease	1	5	6.7%
A – age 65–75 y	1	6–9	>9%
Sex – female	1		

Anticoagulants	Mechanism	Excretion	Dosing Considerations	Risk/Benefit
Warfarin	Vitamin K antagonist	Liver	Adjusted to INR 2–3 Days to therapeutic effect Multiple drug/food interactions (e.g., amiodarone)	Major hemorrhage: 1% per year Intracranial hemorrhage: 0.1–0.6% per year Risk of bleeding increases with INR >3.5 Inexpensive
Dabigatran[b]	Thrombin inhibitor	Kidney		
		CCr >30 mL/min	150 mg bid	Onset of action within hours
		CCr 15–30 mL/min	75 mg bid	No reversal agent for bleeding
			P-glycoprotein substrate (inducers – rifampin, reduce concentration) (inhibitors – amiodarone, verapamil, dronedarone, quinidine), Proton pump inhibitors may reduce absorption	
Rivaroxaban	Xa inhibitor	Kidney	P-glycoprotein substrate	No reversal agent for bleeding
		CCr ≥50 mL/min	20 mg daily	
		CCr 15–50 mL/min	15 mg daily	
Apixaban	Xa inhibitor	Kidney and liver	P-glycoprotein substrate	No reversal agent for bleeding
		Cr >1.5 mg/dL	2.5 mg bid	

✓ As CHAD$_2$ VAS Score increases it increases risk of Stroke

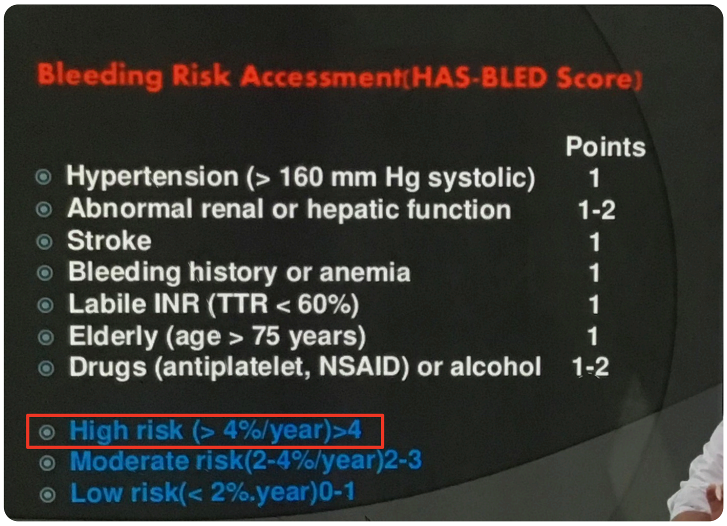
✓ SCORE ZERO : NO ANTICOAGULANT
SCORE ONE : ASPIRIN Alone
SCORE >2 : ORAL ANTICOAGULANT

✓ Pt with score >2
(i) WARFARIN
(ii) DIRECT THROMBIN INHIBITOR : DABIGATRAN - MOST PREFFERED
(iii) ORAL X_A INHIBITOR : RIVAROXABAN
APIXABAN

✓ End-stage CKD not on dialysis : ONLY DRUG WARFARIN

HAS-BLED SCORE

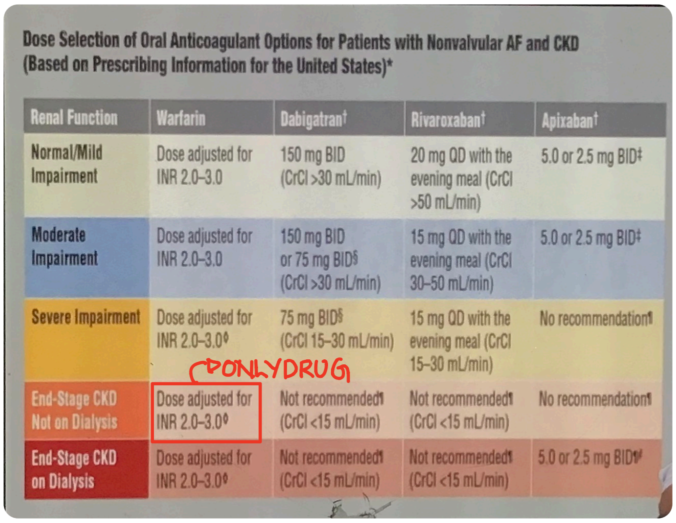
- It determine the BLEEDING RISK after Anticoagulant Therapy



Bleeding Risk Accessment(HAS-BLED Score)

	Points
Hypertension (> 160 mm Hg systolic)	1
Abnormal renal or hepatic function	1-2
Stroke	1
Bleeding history or anemia	1
Labile INR (TTR < 60%)	1
Elderly (age > 75 years)	1
Drugs (antiplatelet, NSAID) or alcohol	1-2

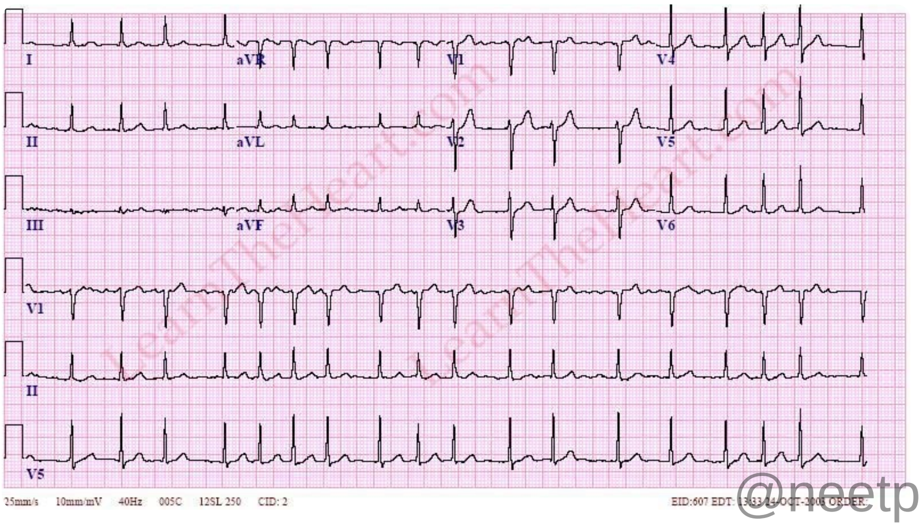
- High risk (> 4%/year)>4
- Moderate risk(2-4%/year)2-3
- Low risk(< 2%.year)0-1



Dose Selection of Oral Anticoagulant Options for Patients with Nonvalvular AF and CKD (Based on Prescribing Information for the United States)*

Renal Function	Warfarin	Dabigatran[†]	Rivaroxaban[†]	Apixaban[†]
Normal/Mild Impairment	Dose adjusted for INR 2.0–3.0	150 mg BID (CrCl >30 mL/min)	20 mg QD with the evening meal (CrCl >50 mL/min)	5.0 or 2.5 mg BID[‡]
Moderate Impairment	Dose adjusted for INR 2.0–3.0	150 mg BID or 75 mg BID[§] (CrCl >30 mL/min)	15 mg QD with the evening meal (CrCl 30–50 mL/min)	5.0 or 2.5 mg BID[‡]
Severe Impairment	Dose adjusted for INR 2.0–3.0[◊]	75 mg BID[§] (CrCl 15–30 mL/min)	15 mg QD with the evening meal (CrCl 15–30 mL/min)	No recommendation[¶]
End-Stage CKD Not on Dialysis	Dose adjusted for INR 2.0–3.0[◊]	Not recommended[¶] (CrCl <15 mL/min)	Not recommended[¶] (CrCl <15 mL/min)	No recommendation[¶]
End-Stage CKD on Dialysis	Dose adjusted for INR 2.0–3.0[◊]	Not recommended[¶] (CrCl <15 mL/min)	Not recommended[¶] (CrCl <15 mL/min)	5.0 or 2.5 mg BID[¶]

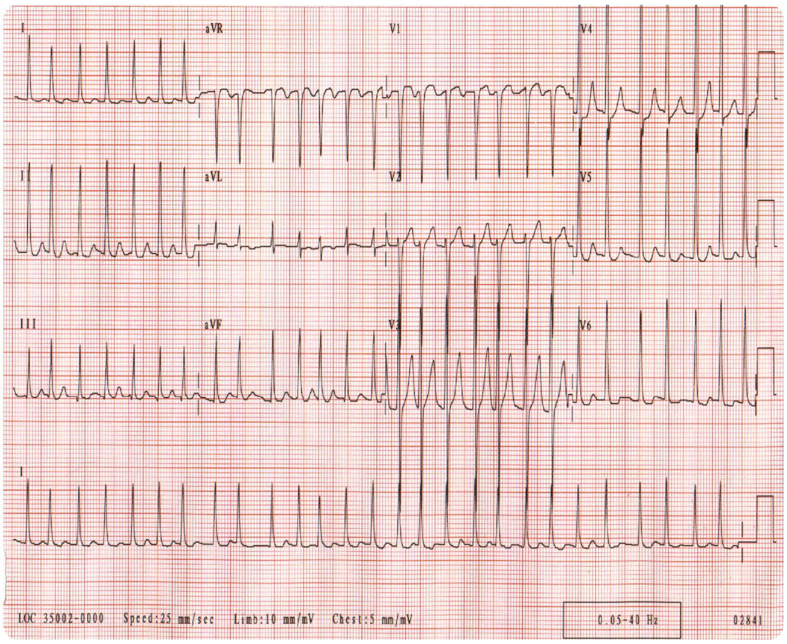
ONLY DRUG


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 40Hz 005C 12SL 250 CID: 2
EID:607 EDT: 13:33 ...-OCT-2005 ORDER:

- NARROW QRS COMPLEX
- IRREGULAR RR INTERVAL
- NOT EVEN A SINGLE MORPHOLOGICALLY IDENTIFIABLE NORMAL P WAVE
- FIBRILLATORY WAVES

↓

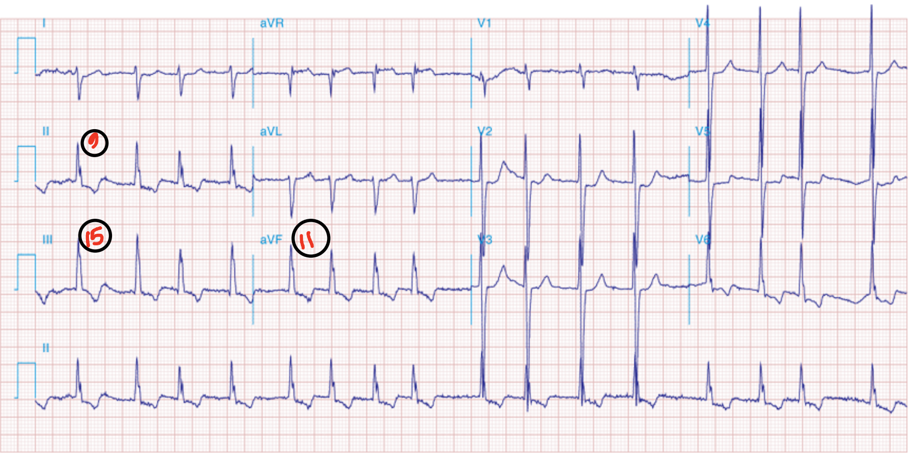
ATRIAL FIBRILLATION



- NARROW QRS COMPLEX
- IRREGULAR RR INTERVAL
- NOT EVEN A SINGLE MORPHOLOGICALLY IDENTIFIABLE NORMAL P WAVE
- FIBRILLATORY WAVES

↓

ATRIAL FIBRILLATION



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- NARROW QRS COMPLEX
- IRREGULAR RR INTERVAL
- NOT EVEN A SINGLE MORPHOLOGICALLY IDENTIFIABLE NORMAL P WAVE
- FIBRILLATORY WAVES

↓

ATRIAL FIBRILLATION

AXIS OF ECG

B/W LEAD III & AVF

↓ ↓

90° - 120°

RAD

MANAGEMENT OF AF

- Rate control & Rhythum control done. Both are having equal efficasy.
- RHYTHUM CONTROL FOR
  1. YOUNG PATIENT with NEW ONSET AF
  2. REVERSIBLE CAUSE FOR AF
  3. CHF precipitated by AF
  4. LONE AF
  5. PATIENT CAN'T TAKE ANTICOAGULANT
- RHYTHUM CONTROL

(A) PATIENT HEAMODYNAMICALLY STABLE

Class I_A : PROCAINIDE
Class I_C : FLECAINIDE
Class III : DOBUTILIDE, IBUTILIDE, AMIODARONE } APPROVED IN PERSISTENT AF
↳ Preffered 1mg i/v for 10 min.

PATIENT WITH CAD/CHF/CCF : AMIODARONE or DOFETILIDE or SOTALOL

(B) PATIENT WITH HEAMODYNAMICALLY UNSTABLE
- TOC : DC Cardioversion : BIPHASIC 200J

↓

if it's planned then give ANTICOAGULANT 3 weeks before

↓

Then do DC Cardioversion

↓

Then give ANTICOAGULANT 4 weeks

- CATHETER ABLATION : DEFINITIVE TREATMENT
- MAZE PROCEDURE : SURGICAL PROCEDURE
- Opening of PULMONARY VEIN is the site which precipitate Lt ATRIUM

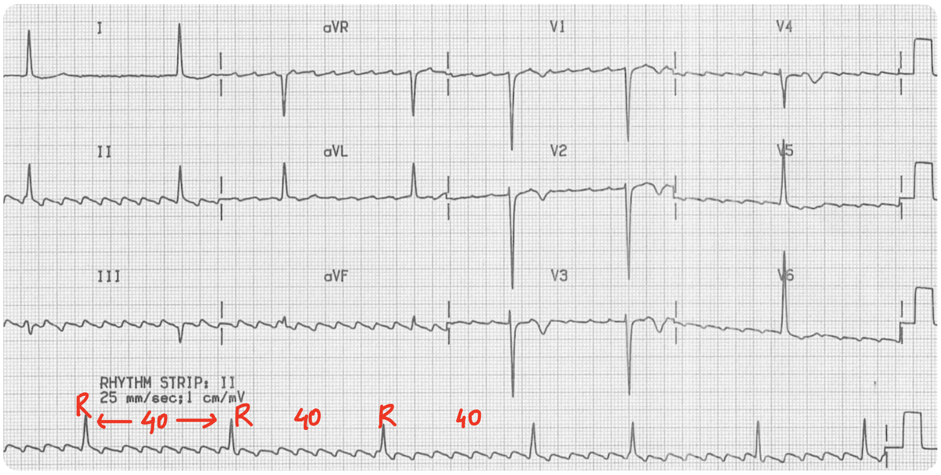
- if PATIENT PRESENTED AFTER 48 HRS

↓

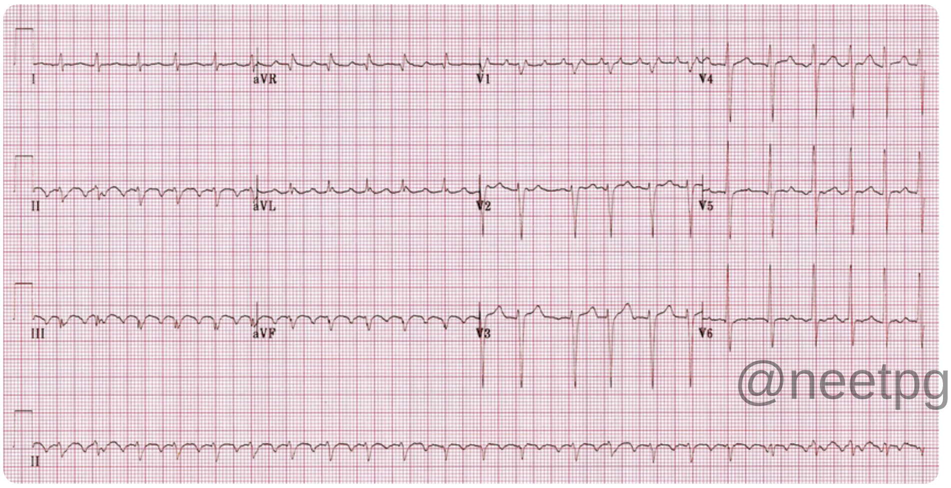
DO TRANSESOPHGEAL ECHO (TEE) to pick up the THROMBUS FORMED

- RATE CONTROL : CALCIUM CHANNEL BLOCKERS : VERAPAMIL 5mg i/v
  BETA BLOCKERS : METOPROLOL (2.5-5 mg i/v)
  DIGOXIN
  AMIODARONE (can also be used)

ATRIAL FLUTTER



- NARROW QRS COMPLEX
- IRREGULAR RR INTERVAL
- FOR ∃ PWAVE one R WAVE
  - PWAVE ↓ inverted
  - ↓ HIGH DEGREE BLOCK
- IRREGULAR BASELINE
- PP interval : 300-350 ms
- UNDULATING BASE LINE + SAW TOOTHED WAVES = ATRIAL FLUTTER with AV Block



- NARROW QRS COMPLEX
- IRREGULAR RR INTERVAL
- UNDULATING BASE LINE
- SAW TOOTHED/FLUTTER WAVE
- INVERTED P WAVE
  INVERTED in LEAD II & +ve in V1
- ATRIAL FLUTTER with Block

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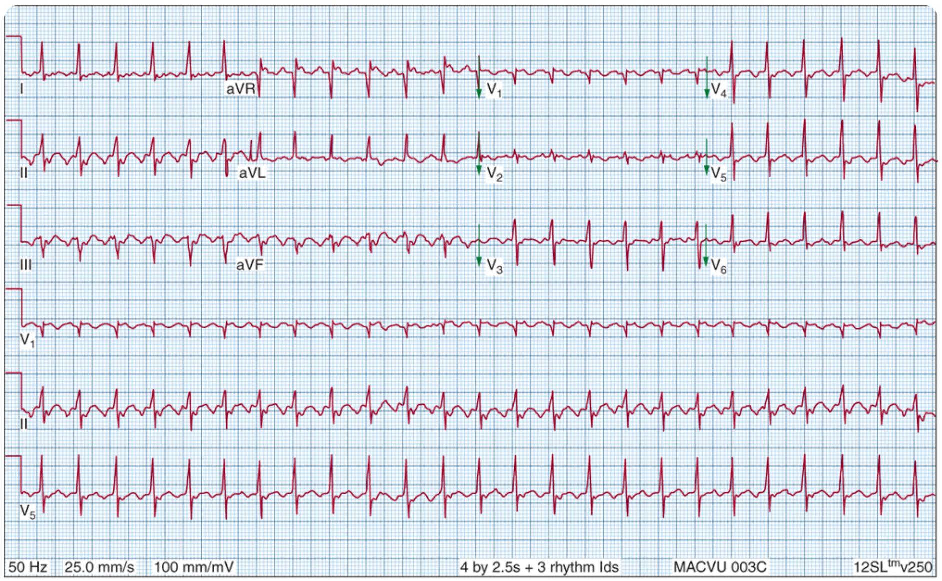
GENERALLY FLUTTER P waves are
POSITIVE IN : V_1
INVERTED IN : LEAD II
} ∵ they are Right sided Flutter & come out through the right wall & go back via Interatrial septum



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- NARROW QRS COMPLEX
- REGULAR RR INTERVAL
- Rate : 150/min
- P waves before QRS Complex with ABNORMAL P WAVE
- UNDULATING BASELINE + POSITIVE PWAVE in V_1 + INVERTED P WAVE in II
- ATRIAL FLUTTER with 2:1 block (as the Atrial rate >300/min it's not Atrial tachycardia)

- SVT Arrhythmia with atrial rate of 250-350/min
- 90% across LATERAL WALL OF RIGHT ATRIUM ∴ P waves are positive in V_1
  P waves are negative in II, III, aVF
- VERY REGULAR UNIFORM FLUTTER WAVES with SAW TOOTHED WAVES
- MC ARRHYTHMIA AFTER OPEN HEART SURGERY : Atrial Flutter (in 1 week)
- ATRIAL COMPLEXES OF : CONSTANT MORPHOLOGY AND CYCLE LENGHTH
  +
  UNDULATING BASELINE

MANAGEMENT

- Risk for Stroke present
- ∴ patient with Post 48 hrs with ATRIAL FLUTTER
  ↓
  ANTICOAGULATE THE PATIENT
- AV Nodal Blockers : β-Blockers, CCB
  ↓
  ↓ impulse coming down to ventricle & make the FLUTTER more PROMINENT
- TOC : DC CARDIOVERSION 25-50J
- IBUTILIDE : MOST EFFECTIVE DRUG IN CONVERTING ATRIAL FIBRILLATION TO NORMAL SINUS RHYTHUM
- SITE OF FOCUS : CAVO TRICUSPID ISTHUMUS

NOTE :

- For atrial Flutter the Atrial rate >300/min. But for AT it's <300/min
- No role for carotid sinus massage as the Mechanism is not RE-ENTRY

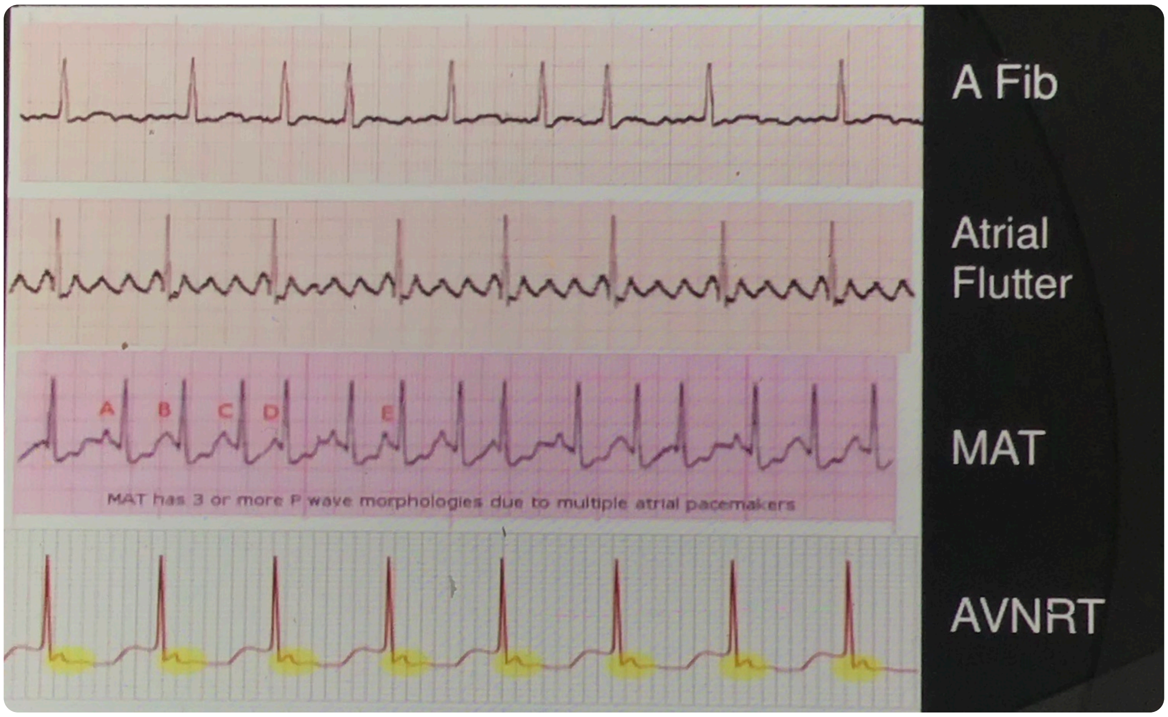
NARROW QRS COMPLEX

REGULAR RR INTERVAL

1. Normal Morphology Pwave : ST
2. Abnormal Morphology Pwave : AT
3. No P Wave : AVNRT / Junctional
4. P wave outside QRS Complex
   - RP > 80 → AVRT
   - RP < 80 → AVNRT
5. UNDULATING BASELINE : AF

IRREGULAR RR INTERVAL

1. NO P WAVE : AF
2. 3 or More different morphology P waves } MAT
3. FLUTTER } with VARIABLE BLOCK
4. AT } with VARIABLE BLOCK



MAT has 3 or more P wave morphologies due to multiple atrial pacemakers

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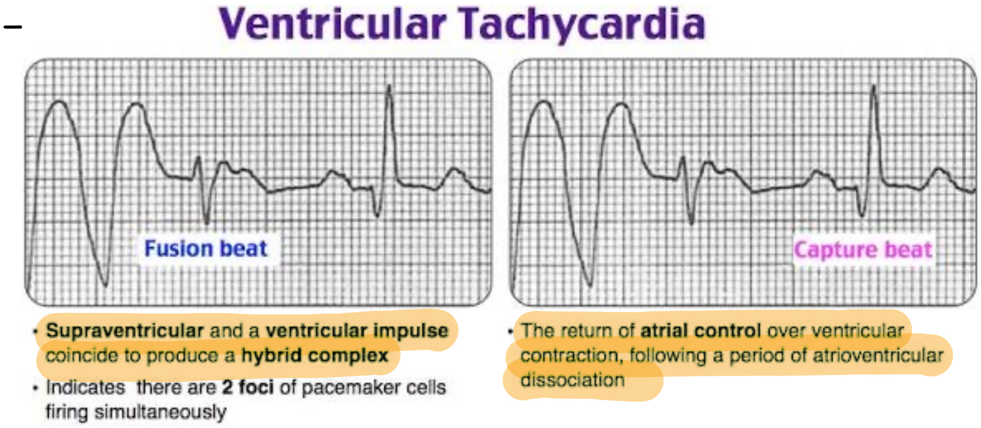
WIDE QRS COMPLEX TACHYCARDIA

✓ Most commonly it's VENTRICULAR TACHYCARDIA (VT)
✓ SUPRAVENTRICULAR IMPULSE with BUNDLE BRANCH BLOCK
✓ ANTIDROMIC AVRT

VENTRICULAR TACHYCARDIA - MONOMORPHIC

- In Ventricular tachycardia, there is AV Dissociation i.e atria & ventricle get seperated completely. (AV ASYNCHRONY)

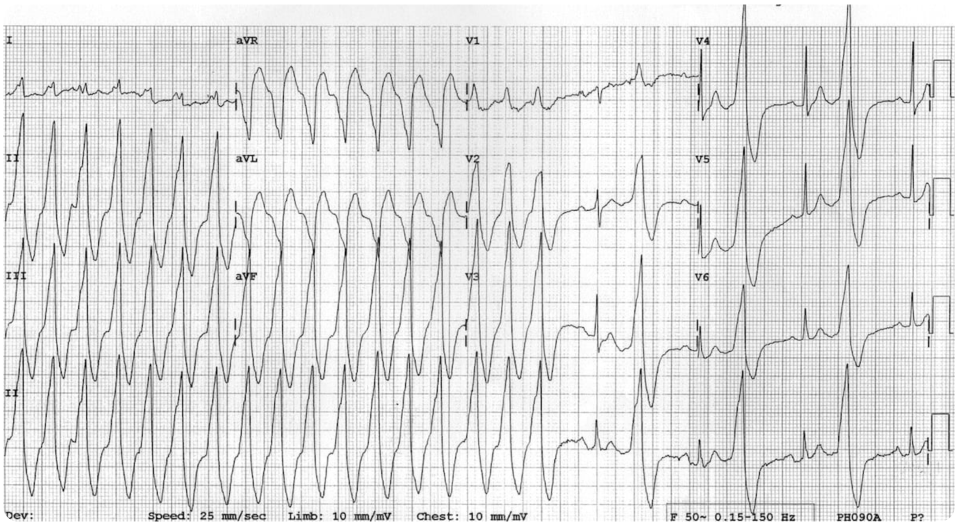
–



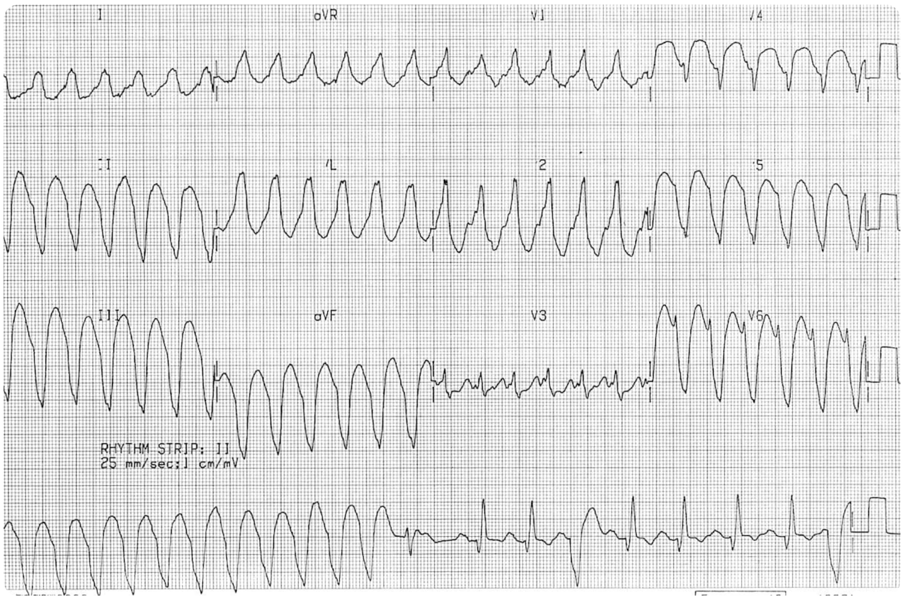
Ventricular Tachycardia

- **Supraventricular** and a **ventricular impulse** coincide to produce a **hybrid complex**
- Indicates there are **2 foci** of pacemaker cells firing simultaneously

- The return of **atrial control** over ventricular contraction, following a period of atrioventricular dissociation



- WIDE QRS COMPLEX
- FUSION BEATS:
  - occurs when a sinus & ventricular beat coincide & produce a hybrid complex
- CAPTURE BEATS:
  - occurs when SAN transiently captures the ventricle, in the midst of AV dissociation to produce Normal DURATION QRS Complex.



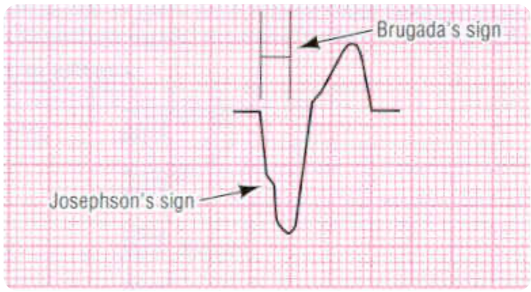
- WIDE QRS COMPLEX
- FUSION BEATS
- CAPTURE BEATS

↓

VENTRICULAR TACHYCARDIA

NOTE: AXIS OVER HERE NORTH WEST as aVR is positive

JOSEPHSON'S SIGN & BRUGADA'S SIGN

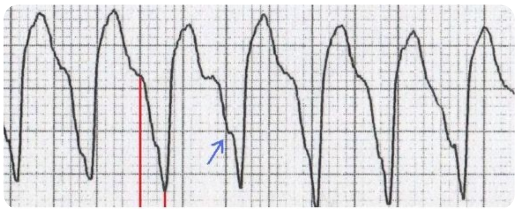


JOSEPHSON'S SIGN:

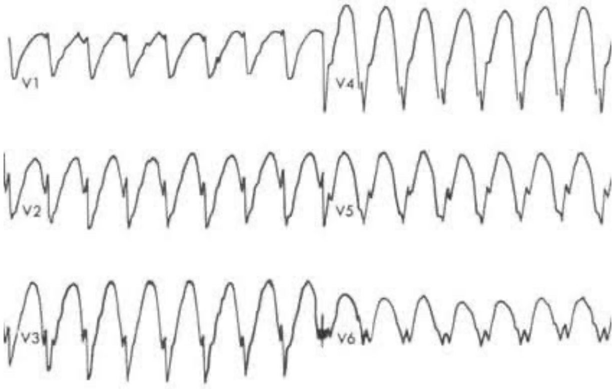
- Notching near the nadir of S-wave

BRUGADA'S SIGN:

- The distance from the onset of QRS Complex to nadir of S wave
- It's >100ms



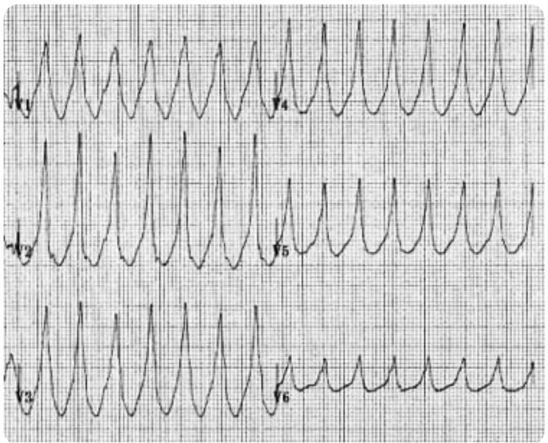
NEGATIVE & POSITIVE CONCORDANCE



↓

NEGATIVE CONCORDANCE

V_1-V_6 contain negative waves



↓

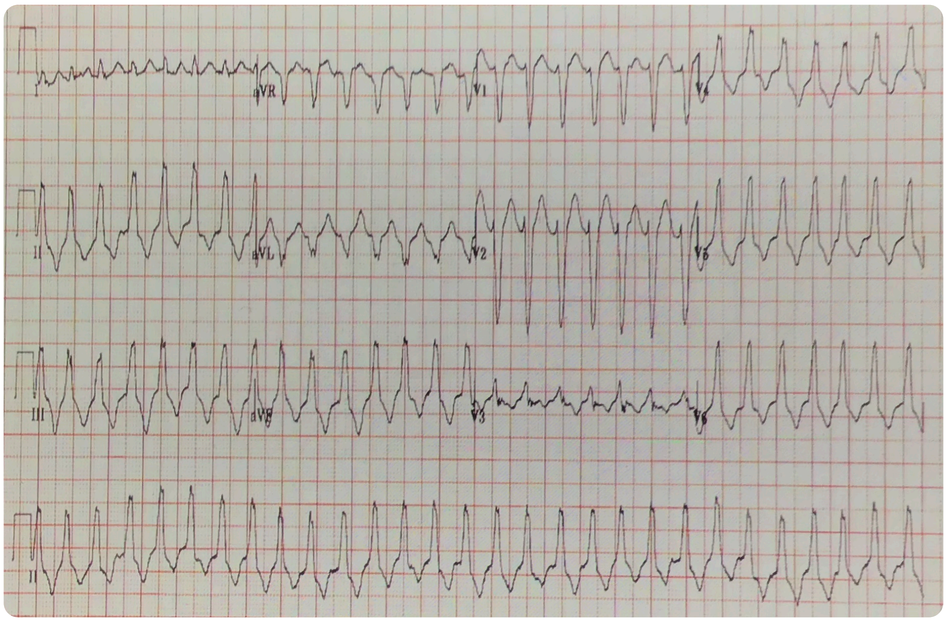
POSITIVE CONCORDANCE

V_1-V_6 contain positive waves

- In VT either positive or negative concordance present.

KEY POINTS OF VT

1. WIDE QRS TACHYCARDIA (>160ms) Rate >100 beats/min
2. POSITIVE/NEGATIVE CONCORDANCE
3. AV DISSOCIATION
4. CAPTURE BEATS
5. BRUGADA'S SIGN
6. JOSEPHSON'S SIGN
7. MOSTLY NORTH-WEST AXIS : I, II, III is inverted
   (Extreme axis deviation) aVR is upright
8. Absence of typical RBBB or LBBB morphology



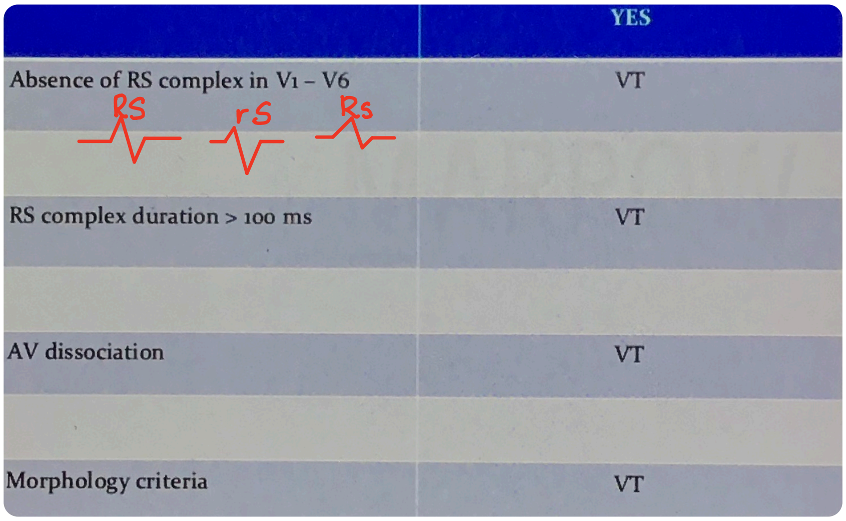
- WIDE QRS COMPLEX
- REGULAR RR INTERVAL
- NO CONCORDANCE
- AV DISSOCIATION

↓

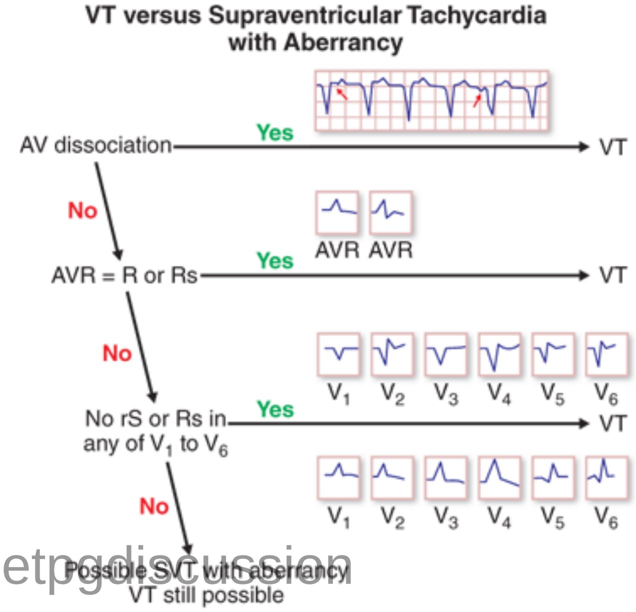
VENTRICULAR TACHYCARDIA

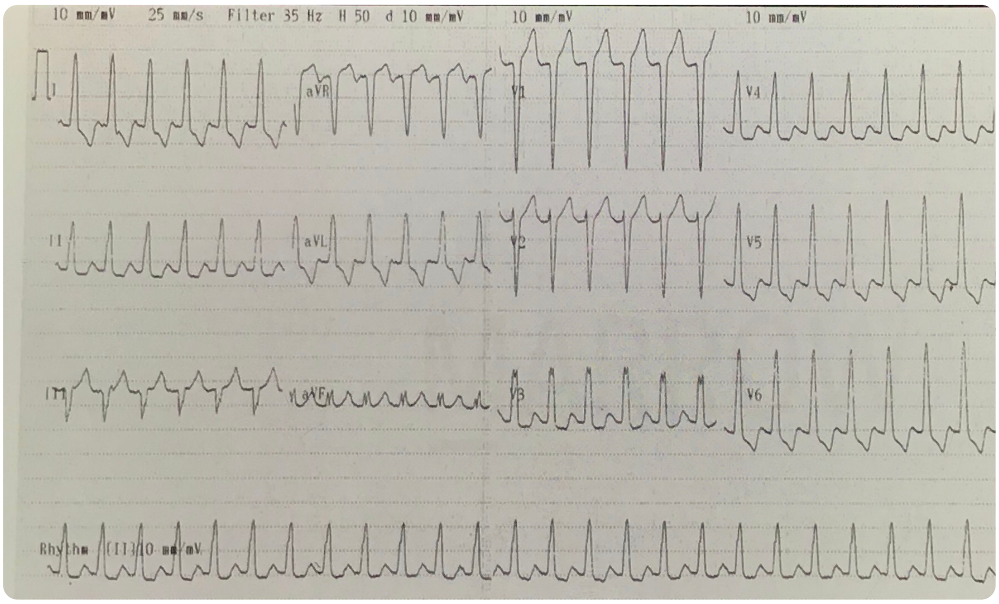
BRUGADA CRITERIA

TO DETERMINE whether it's SVT with aberrancy or VT.



	YES
Absence of RS complex in V1 – V6 RS rS Rs	VT
RS complex duration > 100 ms	VT
AV dissociation	VT
Morphology criteria	VT


VT versus Supraventricular Tachycardia with Aberrancy
AV dissociation
Yes
VT
No
AVR AVR
AVR = R or Rs
Yes
VT
No
V1 V2 V3 V4 V5 V6
No rS or Rs in any of V1 to V6
Yes
VT
V1 V2 V3 V4 V5 V6
No
Possible SVT with aberrancy
VT still possible



Rate >200 beats/min

- NARROW QRS COMPLEX (as VT is >160ms)
- LBBB Morphology
- No P waves

↓

AVNRT with LBBB Morphology

MANAGEMENT OF VT

- Generally Patients are Hemodynamically unstable

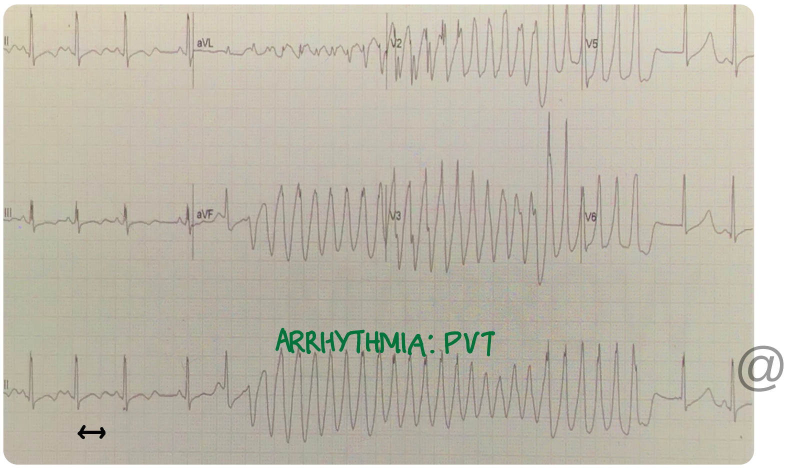
↓

TOC : ∴ Biphasic DC Cardioversion (100-360J) (MC : 200J)

- if patient is HEMODYNAMICALLY STABLE
  - → 1. PROCAINAMIDE in monomorphic VT c̄ good LV function
  - → 2. AMIODARONE in LV Dysfunction
  - → 3. LIGNOCAINE in MI

VENTRICULAR TACHYCARDIA - POLYMORPHIC

- When different QRS Complexes of Different heights : Polymorphic
- Typically associated with TORSADE DE POINTES
- Polymorphic VT + PROLONGED QT INTERVAL = TORSADE DE POINTES



- Prolongation of QT interval

↓

Trigger an arrhythmia (Polymorphic VT)

↓

This is called TORSADE DE POINTES

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CAUSES OF QT PROLONGATION

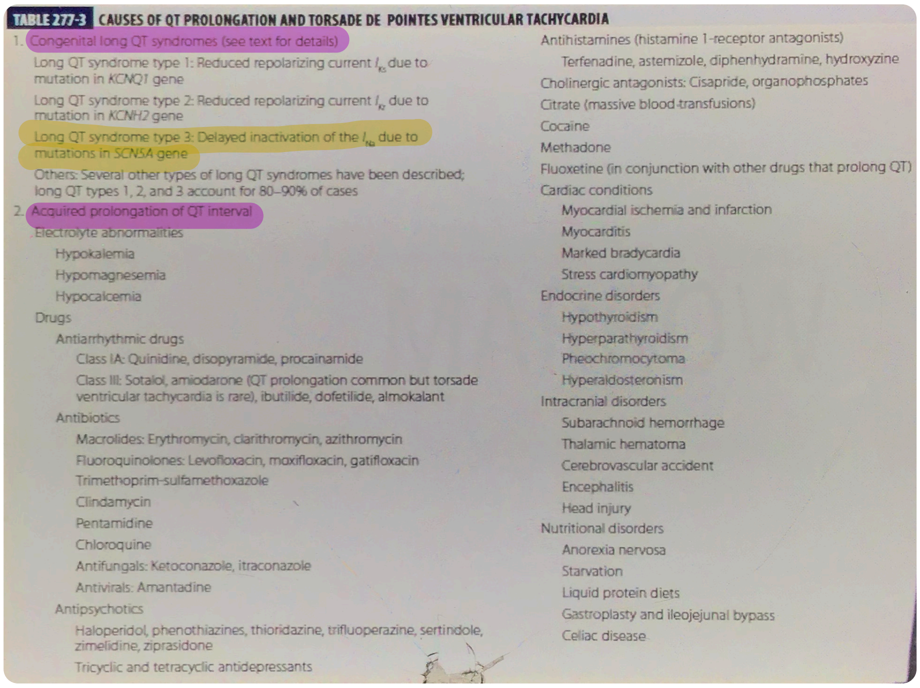


TABLE 277-3 CAUSES OF QT PROLONGATION AND TORSADE DE POINTES VENTRICULAR TACHYCARDIA

1. Congenital long QT syndromes (see text for details)
   - Long QT syndrome type 1: Reduced repolarizing current I_{Ks} due to mutation in *KCNQ1* gene
   - Long QT syndrome type 2: Reduced repolarizing current I_{Kr} due to mutation in *KCNH2* gene
   - Long QT syndrome type 3: Delayed inactivation of the I_{Na} due to mutations in *SCN5A* gene
   - Others: Several other types of long QT syndromes have been described; long QT types 1, 2, and 3 account for 80–90% of cases
2. Acquired prolongation of QT interval
   - Electrolyte abnormalities
     - Hypokalemia
     - Hypomagnesemia
     - Hypocalcemia
   - Drugs
     - Antiarrhythmic drugs
       - Class IA: Quinidine, disopyramide, procainamide
       - Class III: Sotalol, amiodarone (QT prolongation common but torsade ventricular tachycardia is rare), ibutilide, dofetilide, almokalant
     - Antibiotics
       - Macrolides: Erythromycin, clarithromycin, azithromycin
       - Fluoroquinolones: Levofloxacin, moxifloxacin, gatifloxacin
       - Trimethoprim-sulfamethoxazole
       - Clindamycin
       - Pentamidine
       - Chloroquine
       - Antifungals: Ketoconazole, itraconazole
       - Antivirals: Amantadine
     - Antipsychotics
       - Haloperidol, phenothiazines, thioridazine, trifluoperazine, sertindole, zimelidine, ziprasidone
       - Tricyclic and tetracyclic antidepressants
     - Antihistamines (histamine 1-receptor antagonists)
       - Terfenadine, astemizole, diphenhydramine, hydroxyzine
     - Cholinergic antagonists: Cisapride, organophosphates
     - Citrate (massive blood transfusions)
     - Cocaine
     - Methadone
     - Fluoxetine (in conjunction with other drugs that prolong QT)
   - Cardiac conditions
     - Myocardial ischemia and infarction
     - Myocarditis
     - Marked bradycardia
     - Stress cardiomyopathy
   - Endocrine disorders
     - Hypothyroidism
     - Hyperparathyroidism
     - Pheochromocytoma
     - Hyperaldosteronism
   - Intracranial disorders
     - Subarachnoid hemorrhage
     - Thalamic hematoma
     - Cerebrovascular accident
     - Encephalitis
     - Head injury
   - Nutritional disorders
     - Anorexia nervosa
     - Starvation
     - Liquid protein diets
     - Gastroplasty and ileojejunal bypass
     - Celiac disease

-CONGENITAL LONG QT SYN.

a) Long QT Syndrome Type 3: due to Mutation in SCN5A gene ↓ in Brugada syn.

c) Jerwell Lange Niolson Syndrome is associated with sensory neuro hearing loss with QT prolongation.

d) Romanoward Syndrome

- ACQUIRED QT PROLANGATION

a) Most important cause : Drugs : CLASS I_A AAD
CLASS III AAD
ANTIBIOTICS : MACROLIDES, CLINDAMYCIN
ANTIFUNGAL : KETOCONAZOLE
ANTIVIRAL : AMANITIDINE
ANTIPSYCHOTICS : HALOPERIDOL, TCAs
ANTI HISTAMINE : TERFENADINE, ASTEMIZOLE, FEXOFENADINE

b) CARDIAC CONDITIONS : MI

c) ENDOCRINE DISORDER : HYPOTHYROIDISM
HYPER PARATHYROIDISM

d) INTRACRANIAL DISORDER : CNS BLEED/INFARCTION

e) ELECTROLYTE ABNORMALITY : HYPO Ca^{2+}
HYPO K^+
HYPO Mg^2

f) HYPOTHERMIA

NOTE:

HYPERCALCEMIA
DIGOXIN
HYPERTHERMIA
} SHORTEN QT INTERVAL

TREATMENT OF POLYMORPHIC VT

- Mostly patients are HEMODYNAMICALLY UNSTABLE
  TOC : DEFIBRILLATION

✓ if patient is hemodynamically STABLE & ACQUIRED QT SYNDROME

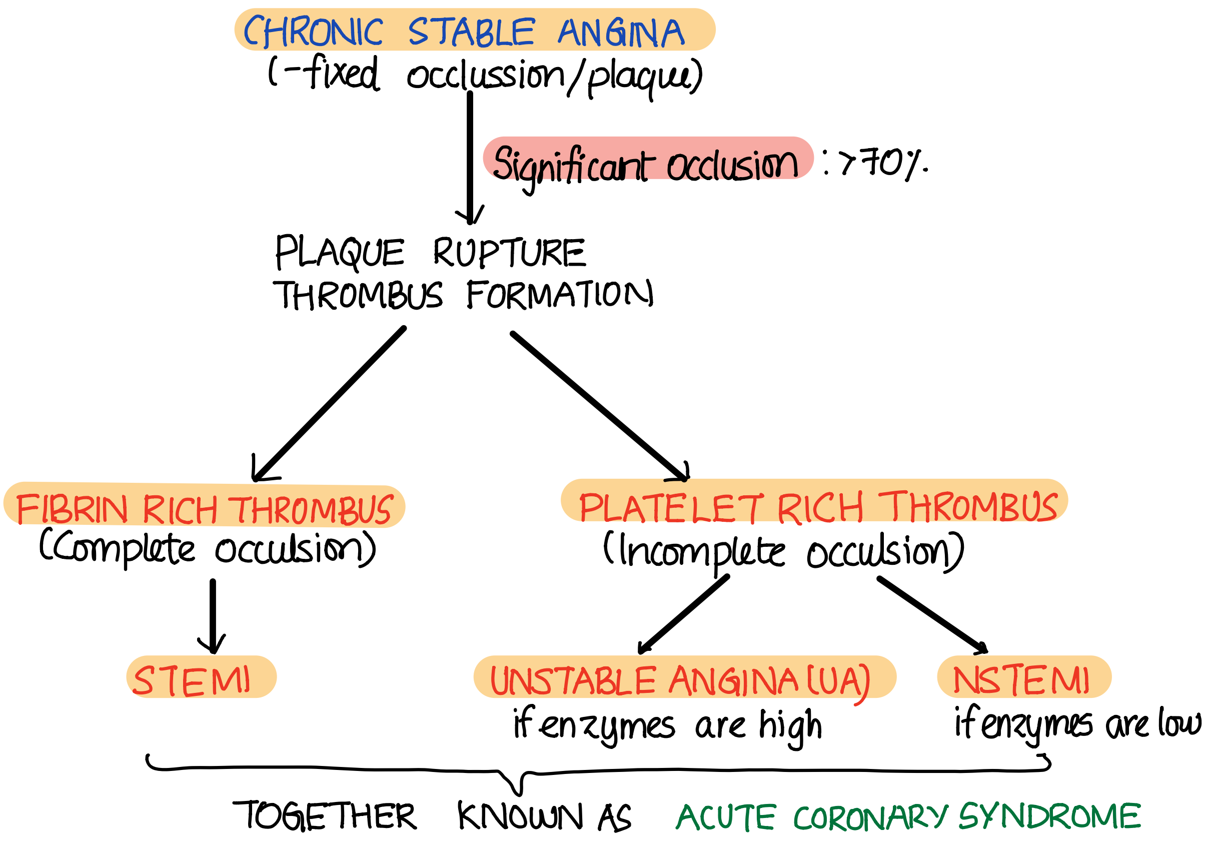
i/v Mg^{2+} $\xrightarrow[\text{Then}]{}$ TEMPORARY PACING

– As it can reccur : IMPLANTABLE CARIOVERTOR DEFRILLATOR

✓ if patient is hemodynamically STABLE & CONGENITAL QT SYNDROME
DOC : BETA BLOCKERS

PART – 1

ACUTE CORONARY SYNDROME



CHRONIC STABLE ANGINA
(-fixed occlussion/plaque)

↓ Significant occlusion : >70%

PLAQUE RUPTURE
THROMBUS FORMATION

- FIBRIN RICH THROMBUS (Complete occulsion) → STEMI
- PLATELET RICH THROMBUS (Incomplete occulsion)
  - UNSTABLE ANGINA (UA) – if enzymes are high
  - NSTEMI – if enzymes are low

TOGETHER KNOWN AS ACUTE CORONARY SYNDROME

2/3 CRITERIA FOR ACS : 2 out of 3 Criterias are mandatory to diagnose ACS

① SYMPTOMS ② ECG ③ ENZYME

AIMS

1. TO PICK UP PATIENT WITH CHRONIC STABLE ANGINA OR HIGH RISK PATIENTS (Eg: DM)

↓ go for

INDUCIBLE ISCHAEMIA
(∵ Base line ECG is Normal)
→ TMT / Stress ECG
→ Perfusion Scintigraphy

↓ do

ANGIOGRAPHY

↓ come out with

Angioplasty / CABG

2. TO PICK PATIET WITH ACUTE ACS —CLASSIFY→
   - STEMI
   - NSTEMI
   - UA

RISK FACTORS

- Family history of MI
- Chronic Stable Angina : Angina on exertion (or) Dyspnea on Exertion
- CIGARETTE SMOKING : MOST CRUCIAL YET PREVENTABLE RISK FACTOR

  ↓ do a Angiogram
  Caliber of vessal is lost
  ∴ DISTAL TAPPERING of BV
  (You can't stent the vessal)

  * Smoking 5-6 packet/day for 20 years (or)
  2-3 packet/day for 10-12 years
  * Also Diffuse Vascular Disease present.
  As it effect
    - ILLIAC VESSAL
    - LOWER LIMB VESSAL
    - RENAL ARTERY
    - CORONARY ARTERY
- HYPERTENSION (BP ≥ 140/90)
- HIGH LDL

(i) Highly Sensitive CRP : DFFINITE MARKER OF ATHEROSCLEROSIS
→ Lp(a) Level also
(iii) Fibrinogen level
(v) TG/HDL Ratio

(ii) ApoB100/HDL Ratio
(iv) Total Cholesterol/HDL Ratio
(vi) LDL/HDL Ratio

- LOW HDL (Most important : HDL-2)
- DIABETES MELLITUS
  * leads to INSULIN RESISTENCE
- IN WORLD MALES HAVING MORE RISK THAN FEMALE
  → Male > 45 yrs
  Female > 55 yrs

  IN INDIA
    - Age of presentation : 45-50 YRS
    - FEMALE > MALE

  FEMALE ↓ ACS present as ATYPICAL PRESENTATION
  MALE ↓ ACS present as CHEST PAIN
- OBESITY
  But IN INDIA : Even Pt with NORMAL BMI : Risk of ACS
- MALNUTRITION, INFLAMMATION, ATHEROSCLEROSIS
- HOMO CYSTENEMIA
- METABOLIC SYNDROME

  Waist Circumference : 120/88
  TG : 150
  BP : 130/85
  FBP : 100
  HDL : <50/<40

✓ All these patient should go for → STRESS ECG
→ PERFUSION SCINTIGRAPHY

↓

not for Resting ECG : It's normal in these patient
(or) Echocardiogram : Structurally the heart is normal
Sometimes Diastolic dysfunction in some patient

✓ 5-6 patients out of 10 ACS patient develop Heart failure because of INFARCTION
This is called as ISCHAEMIC DIALATED CARDIOMYOPATHY

INDICATIONS TO PUT PATIENTS ON STATINS

1. Clinical Atherosclerosis evidence
2. DM (40-75 yrs age)
3. LDL >100 mg/dl

} MODERATE TO HIGH DOSE STATIN
40mg ATORVASTATIN (or)
20mg ROSUVASTATIN

- AS STATIN : NO EFFECT ON HDL
NO EFFECT ON TG
ONLY EFFECT ON LDL
- MOST Patients have DYBETIC DYS LIPIDEMIA
due to ↓HDL
↑TG
↑ small dense/oxidised LDL → MOST ATHEROGENIC

} NO EFFECT BY STATINS
↳ ∴ SAROGLITAZAR

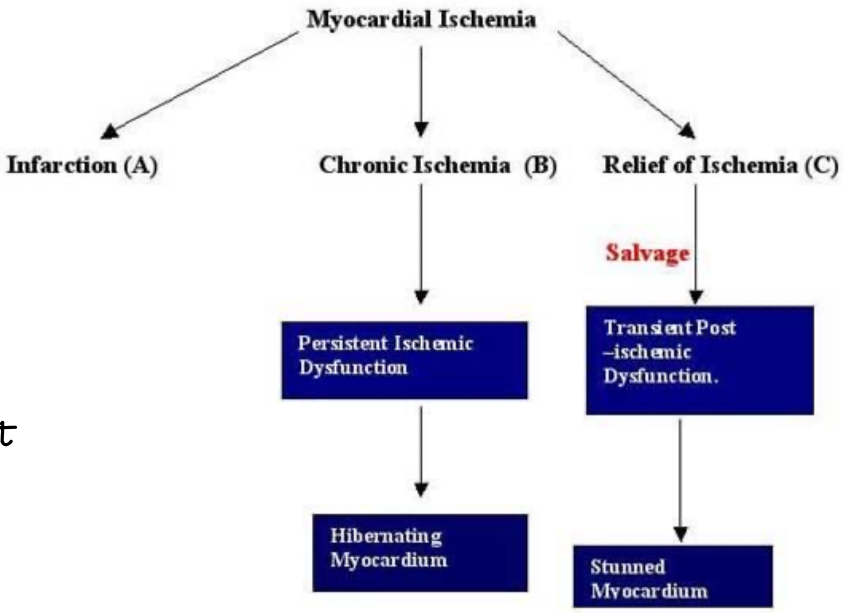
HIBERNATING MYOCARDIUM & STUNNED MYOCARDIUM

HIBERNATING MYOCARDIUM

- Areas of myocardium which are persistently under perfused but still viable after reperfusion
- Occurs in chronic ischaemia
- Reversible

STUNNED MYOCARDIUM

- Segmental dysfunction which persist for a variable period of time after reperfusion is called STUNNING
- Occurs in Acute ischaemia.
- Reversible



NOTE:

- HOPE Trial Demonstrates that RAMIPRIL reduces fatal & non fatal vascular events in high risk patients

- On Target study reveals that ARBs are not inferior to ACE ⊖

↓ use of ARB
⊖ AT1 Ⓡ ⟶ Upregulate AT2 Ⓡ ⟶ CAUSE Malignant Changes
Eg: Eosophageal malignancy
Pharyngeal/
Laryngeal malignancy } with CADESARTAN

- CONTRAINDICATIONS FOR ACE ⊖
  1. PREGNANCY
  2. h/o HEREDITARY ANGIONEUROTIC EDEMA
  3. b/l Renal Artery Stenosis

  Note : CREATININE Level is not a C/I

- AFTER starting ACE ⊖ Monitor for Creatinine level
  ① if >30% increase in creatinine level after starting ACE ⊖ } STOP ACE ⊖
  ②. HYPERKALEMIA : if it's > 5.5 –6 within 1wk. ∴ STOP ACE ⊖

DIETERY/LIFESTYLE RECOMMENDATION

- Saturated Fat <7%
- Dietary Cholesterol of < 200mg/day
- Soluble fibre >10–25g/day
- Plant sterols : 2g/day
- >15% protein
- Salt less than 6g/day
- No trans fattyacids

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PART – II

ACUTE CORONARY SYNDROME

SPECTRUM OF PATIENTS

1. HEART FAILURE : → Systolic HF (Most often by patient who are already developed MI) → Diastolic HF
2. CHRONIC STABLE ANGINA : Occurs during exertion <20 minute duration
3. Unstable Angina / NSTEMI : Occurs at Rest, duration >20 minutes
4. STEMI
5. PRINZMETAL'S ANGINA / VASOSPASTIC ANGINA : Occurs at rest <20 minutes duration

CHRONIC STABLE ANGINA

- Defined by
  i) Precordial pain precipitated by stress or exertion
  No pain at rest
  May be tightness / burning / squeezing
  Behind or to left of midsternum with radiation to any dermatome from C8 to T4

  ii) Duration less than 20mnts . . .
  If more than 30mnts suspect ACS

  iii) Excellent response to NTG

- ACUTE MR in chronic stable angina : Diagnosis : Pappilary Muscle dysfunction

EXERCISE ECG / STRESS ECG / TREAD MILL TEST

- Recommended initial procedure
- It's according to BRUCE PROTOCOL
- C/I : * Rest Angina
  * Symptomatic Aortic stenosis
  * Pt who donot want to do the test : DON'T PUSH.
- Significant ST Down slopping : * 2mm in 6mnts before achieving the max HR are considered to be very significant
  * if we keep the cut-off as 1mm : 30% chance of false positivity

MYOCARDIAL PERFUSION SCINTIGRAPHY

- It's also called called Perfusion scan
  ↓
  USE : Thallium
- INDICATION :
  - * Symptoms at Rest
  - * Positive test in asymptomatic patient
- ADVANTAGE
  - * Localize region of ischaemia
  - * Differentiate Ischaemia & Infarction
  - * Acess completeness of revascularisation
- ALTERNATIVE : Exercise Radionucliide Angiography / MUGA Scan

* Perfusion Scintigraphy / MRI can distinguish stunned myocardium from Scarred Myocardium

* Electron bheam CT (EBCT) TO QUANTIFY CARDIAC CALCIFICATION

* MRI : Test of Choice to IDENTIFY MYOCARDIAL FIBROSIS

* AGATSTON SCORE : IS BASED ON EXTENT ON CORONARY ARTERY CALCIFICATION

* 70% or MORE narrowing associated with ischaemia
  50% or More is significant

* INTRAVASCULAR ULTRASOUND : Test of Choice for Ostial left main lesion & coronary dissection

MANAGEMENT

Oral ISDN : Take 30 min
Oral ISMN : Take 45 min

- S/L NTG : Action within 2-5 mints.
  0.3 - 0.6 mg
  3 tablets within 20 min
  ↓ Not Responding
  UA / NSTEMI / STEMI
- Nitrate Tolerance is the main limitation of NTG
  → To prevent : keep Nitrate free interval 8-12 hrs period daily
- Beta Blockers : Antianginal drug with mortality benefit
- Ranolazine : PROLONG EXERCISE DURATION & TIME TO ANGINA
  → S/E : QT Prolongation

- IVABRADINE : Blocks Funny current ⟶ ↓H.R

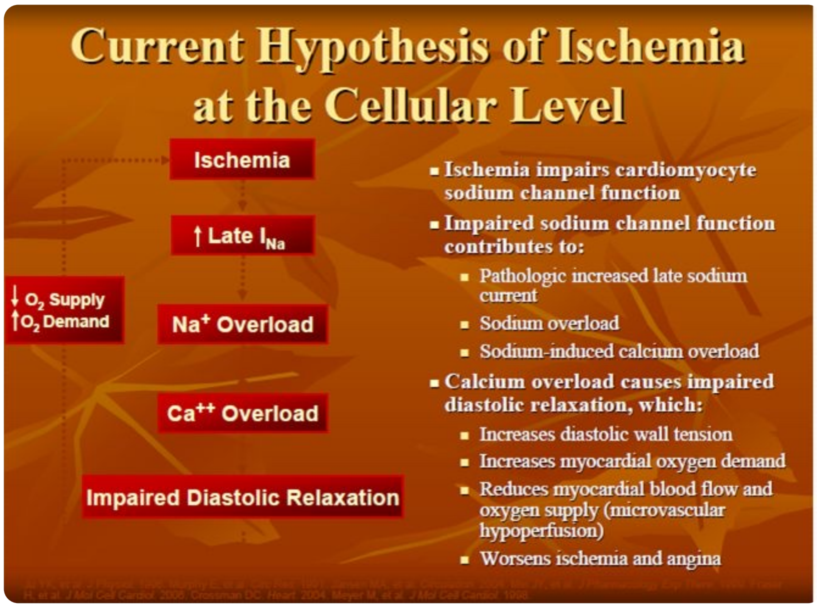
- CCBs are third line drugs

DRUGS IN ANGINA

* First Line : NTG

* Second Line : Beta blockers
Ranolazine
Ivabradine
Nicorandil

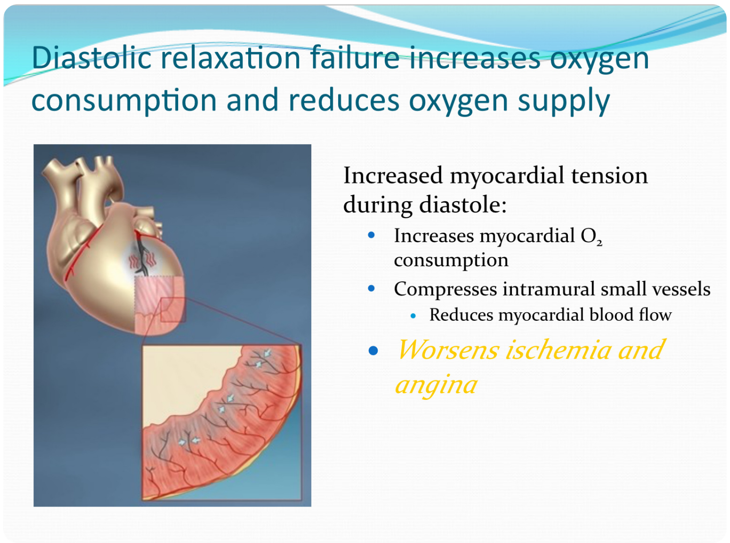
* Third line : CCBs
Trimetazidine



Current Hypothesis of Ischemia at the Cellular Level

- Ischemia impairs cardiomyocyte sodium channel function
- Impaired sodium channel function contributes to:
  - Pathologic increased late sodium current
  - Sodium overload
  - Sodium-induced calcium overload
- Calcium overload causes impaired diastolic relaxation, which:
  - Increases diastolic wall tension
  - Increases myocardial oxygen demand
  - Reduces myocardial blood flow and oxygen supply (microvascular hypoperfusion)
  - Worsens ischemia and angina

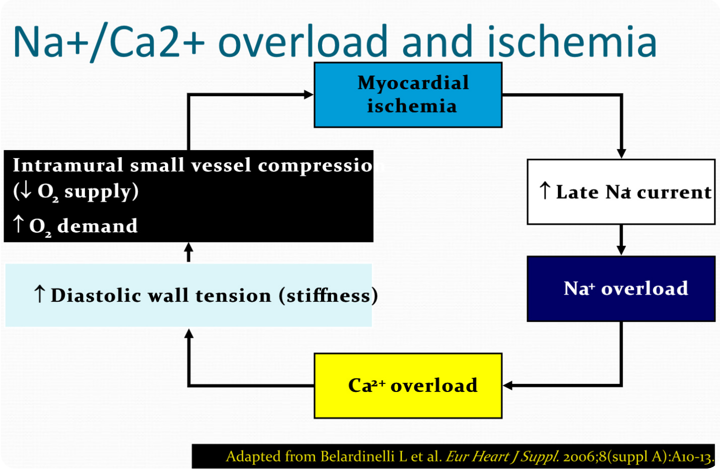
ISCHAEMIA
↓
Malfunction of Na^+ Channel
↓
↑ Late Sodium Current
↓
Na^+-induced Ca^{2+} Overload
↓
Impaired diastolic filling
↓
↑ Myocardial O_2 level



Diastolic relaxation failure increases oxygen consumption and reduces oxygen supply

Increased myocardial tension during diastole:

- Increases myocardial O_2 consumption
- Compresses intramural small vessels
  - Reduces myocardial blood flow
- *Worsens ischemia and angina*



Na+/Ca2+ overload and ischemia

Adapted from Belardinelli L et al. *Eur Heart J Suppl.* 2006;8(suppl A):A10-13.

Reduce E.D.V ⟹ ↓ O_2 Consumption : Nitrate
↓ H.R ⟹ ↓ O_2 Consumption : β⊖, CCBs
pFOX ⊖ ⟹ ↓ O_2 Consumption : Ranolazine
Rhokinase ⊖ ⟹ ↓ O_2 Consumption : Fasudil
SAN ⊖ ⟹ ↓ O_2 Consumption : Ivabradine

TRIMETAZIDINE / RANOLAZINE

- These are p-FOX ⊖

↓

⊖ β oxidation of FFA (↑ATP utilisation)

↓

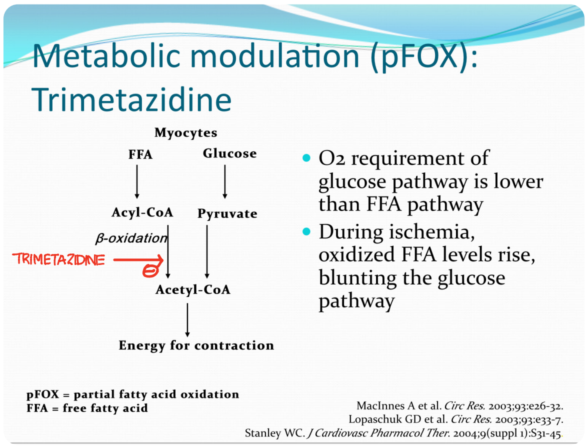
Myocyte utilise the Glucose (↓ATP utilisatn)

↓

↓O_2 Requirement.

- At the time of ishaemia use FA oxidation

RANOLAZINE ↓ S/E: QT Prolongation



Metabolic modulation (pFOX): Trimetazidine

Myocytes
FFA
Glucose
Acyl-CoA
Pyruvate
β-oxidation
TRIMETAZIDINE
⊖
Acetyl-CoA
Energy for contraction

- O_2 requirement of glucose pathway is lower than FFA pathway
- During ischemia, oxidized FFA levels rise, blunting the glucose pathway

pFOX = partial fatty acid oxidation
FFA = free fatty acid

MacInnes A et al. *Circ Res.* 2003;93:e26-32.
Lopaschuk GD et al. *Circ Res.* 2003;93:e33-7.
Stanley WC. *J Cardiovasc Pharmacol Ther.* 2004;9(suppl 1):S31-45.

IVABRADINE

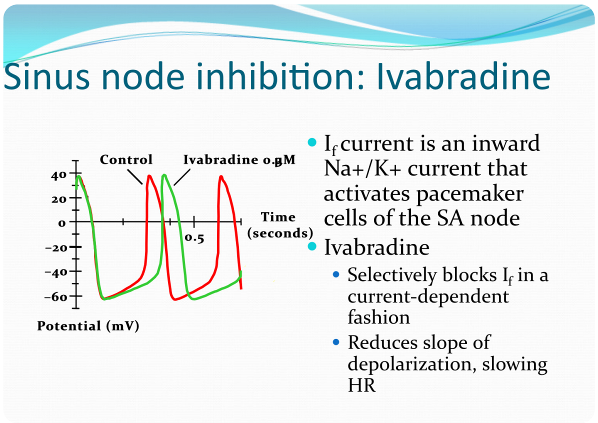
- M.O.A : I_F current ⊖ (funny current)

↓

⊖ SA Node

↓

↓ H.R



Sinus node inhibition: Ivabradine

Control
Ivabradine 0.3 μM
40
20
0
-20
-40
-60
0.5
Time (seconds)
Potential (mV)

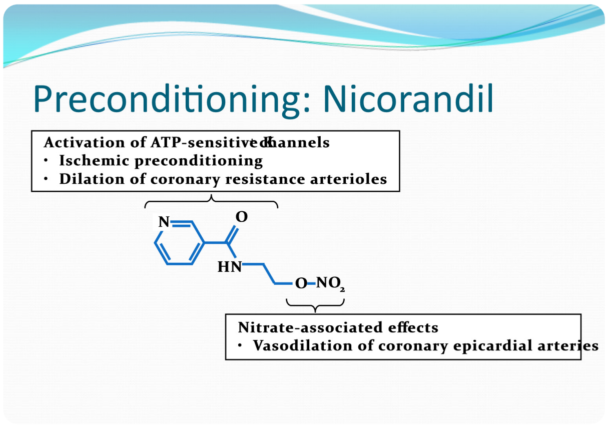
- I_f current is an inward Na+/K+ current that activates pacemaker cells of the SA node
- Ivabradine
  - Selectively blocks I_f in a current-dependent fashion
  - Reduces slope of depolarization, slowing HR

NICORANDIL

- M.O.A : ACTIVATION OF ATP SENSITIVE K^+ CHANNELS

↓

Dialatation of Resistance Vessals



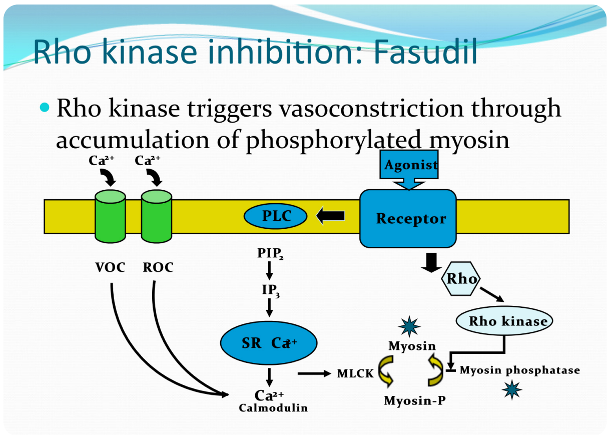
Preconditioning: Nicorandil

Activation of ATP-sensitive K channels
- Ischemic preconditioning
- Dilation of coronary resistance arterioles

Nitrate-associated effects
- Vasodilation of coronary epicardial arteries

FASUDIL

M.O.A : Rho Kinase Inhibitor



Rho kinase inhibition: Fasudil

- Rho kinase triggers vasoconstriction through accumulation of phosphorylated myosin

Ca2+
Ca2+
Agonist
PLC
Receptor
VOC
ROC
PIP2
IP3
SR Ca2+
Ca2+ Calmodulin
MLCK
Rho
Rho kinase
Myosin
Myosin phosphatase
Myosin-P

Summary

Medication Class	Impact on HR	Impact on BP	Physiologic Mechanism
Beta Blockers	↓	↓	**Decreasepump function**
Calc Channel Blockers	↓	↓	**DecreasePump function+ Vaso-dilitation**
Nitrates	Reflux tachy cardia ↑	↓	**Vaso-dilitation**
Ranolazine	O	O	Reduced Cardiac Stiffness

Q : Patient with Precordial chest pain during rest without precipitant rapidly relieved by Nitrates , Transient ST Elevation on ECG. Normal Angiography. Nitrate/ CCB useful but not preffered

* chronic pattern of recurrent episodes of chest pain
* Episodes midnight to early morning.
* Episode last for 5 to 15 minutes
* Physical Examination : No finding charachteristic to VA. However during episode Tachycardia, HTn, Diaphoresis & a gallop rhythum present

Diagnosis : • PRINZMETAL'S ANGINA / VASOSPASTIC ANGINA . (due to transient vasospastic episodes)

• Seen in smokers • Good Prognosis

ACUTE CORONARY SYNDROME

NSTEMI

- DEFINITION : Rest angina >20 minutes
New ONSET SEVERE ANGINA
CRESCENDO PATTERN

↙ if this patient have
* ST Depression
* Enzymes +
↓
NSTEMI

↘ If this patient have
* ST depression
* Enzymes normal
↓
UNSTABLE ANGINA

- According 2017 classification both together called as NSTE- ACS

- TIMI RISK SCORE : Determine mortality risk in NSTEMI

MANAGEMENT

1. S/L NTG : 0.3- 0.6 mg x 3 times within 20 minutes
↓ NOT RESPONDED
Give MORPHINE

2. ASPIRIN : - Essential drug in ACS; STEMI & NSTEMI
- For 1° PCI or URGENT setting
Loading dose : 325 mg/day (non-enteric coated)
- HARRISON : Hospital dose : 80- 325 mg/day
- Higher doses ↑ bleeding risk without additional benefit
- Every cases of CAD without C/I should be prescribed ASPIRIN
- Maintenance Dose : 75 mg

3. IN EVERY CASE OF NSTE-ACS : Should combine
CLOPIDOGREL X ASPIRIN
↓
Continued for 12 months
Aspirin maintence dose <100mg

Continue ASPIRIN for Life
CLOPIDOGREL for 12 months

Aspirin x ADP Receptor Antagonist
↓
1. CLOPIDOGREL : Loading dose : 300mg
Maintenance : 75 mg
For 1yr (12 month)

→ Drawback :
- Show CYP2C19 Polymorphism
↳ Activator of Clopidogrel

↓
2. PRASUGREL : - 10 time more potent
- Rapid onset of action
- Better absorption
- But still CYP2C19 activation

↓
3. TICAGRELOR
- LD : 180mg
- MD : 90mg BD
- for 12 months

- No activation by CYP2C19
- Reversible inhibitor
- Short t ½ + ORAL
- S/E : Mild Dyspnoea

4. STATINS : - In First 24hrs
- Risk for UA in the next 4 month reduces by 25%

Atorvastatin : 40-80mg

5. $GP_{IIB-IIIA}$ ANTAGONIST :

ABCIXIMAB : PLASMA t½ Short ; Platelet bound t½ : Long
SAFE IN RENAL FAILURE
EPTIFIBATIDE : LONGEST T½
TIROFIBAN

→ ROLE : ↓ PERIPROCEDURAL MI (MI developed during PCI)

6. HEPARIN : - 33% Reduction in death in 1st week
- Major Reduction in non fatal MI
- NO SIGNIFICANT INCREASE IN BLEEDING
- NO DECREASE IN RECURRENT ANGINA OR REVASCULARISATION PROCEDURE

DRUGS : UNFRACTIONATED HEPARIN :
LMWH
FONDAPARINUX
DIRECT THROMBIN ⊖ : Eg : DABIGATRAN

ENOXAPARIN & FONDAPARINUX is preffered

ENOXAPARIN (LMWH) : Advantages over Unfrac. Heparin

↓

for 7-8 days

1. No inhibition of platelet function
2. No Thrombocytopenia
3. NO LIVER TOXICITY
4. Patient variability less

✓ CLASS I INDICATION: ASPIRIN
CLOPIDOGREL
UFH / ENOXAPARIN

✓ • For initial conservative strategy
• to prevent Periprocedural MI
• Indication if pt had Recurrent Angina
• ABCIXIMAB should not be used

} EPTIFIBATIDE
TIROFIBAN } $GP_{IIB\text{-}IIIA}$ ⊖

KILLIP CLASSIFICATION

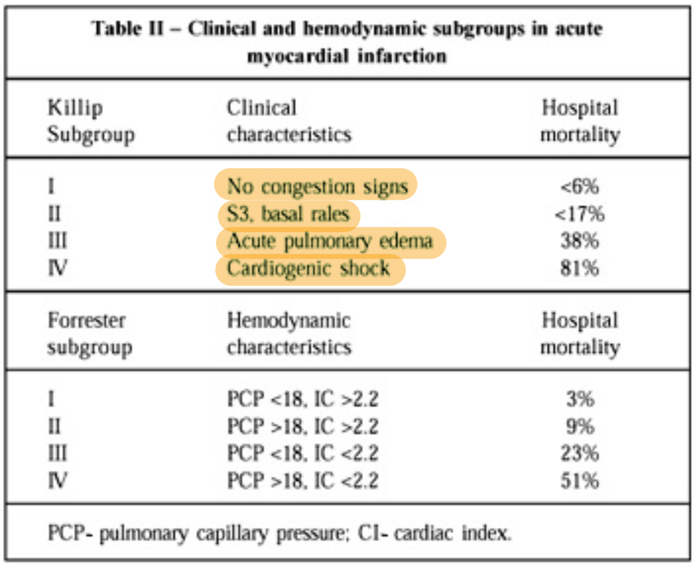


Table II – Clinical and hemodynamic subgroups in acute myocardial infarction

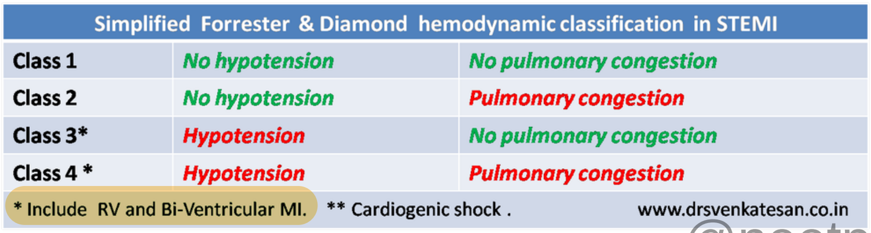
Killip Subgroup	Clinical characteristics	Hospital mortality
I	No congestion signs	<6%
II	S3, basal rales	<17%
III	Acute pulmonary edema	38%
IV	Cardiogenic shock	81%
Forrester subgroup	**Hemodynamic characteristics**	**Hospital mortality**
I	PCP <18, IC >2.2	3%
II	PCP >18, IC >2.2	9%
III	PCP <18, IC <2.2	23%
IV	PCP >18, IC <2.2	51%

PCP- pulmonary capillary pressure; CI- cardiac index.

- Predicting mortality by acessing LV function
- CLASS I : NO SIGN OF LV FAILURE
- CLASS II : S3, BASAL RALES
- CLASS III : PUL. EDEMA
- CLASS IV : CARDIOGENIC SHOCK
- IN LV Failure take to IABP (Intra aortic Balloon Pump)
↓ followed by
Angiogram
- JVP Elevation
Clear Lungs
Hypotention } CASE OF RV FAILURE
↓ t/t
Pump with fluids

FORRESTER & DIAMOND CLASSIFICATION

* It's modified Killip classification



Simplified Forrester & Diamond hemodynamic classification in STEMI

Class 1	No hypotension	No pulmonary congestion
Class 2	No hypotension	Pulmonary congestion
Class 3*	Hypotension	No pulmonary congestion
Class 4 *	Hypotension	Pulmonary congestion

* Include RV and Bi-Ventricular MI. ** Cardiogenic shock . www.drsvenkatesan.co.in

Q Tripple Vessel disease
Severe LV Systolic Dysfunction
2 vessel disease with Proximal LAD occlusion
Left main disease
} Diagnosis : CABG

ACUTE MI

- Evidence of Myocardial necrosis in the settings of Ischaemia

↓ i.e

Rise of cardiac biomarkers (Mostly Troponin)
followed by any of the following

1. New ST/T Changes or New LBBB
2. Symptoms of ischaemia
3. Pathological Q waves on ECG
4. Evidence of thrombus by angiogram
5. Impaired perfusion on scintigraphy or new Regional Wall Motion anomaly on Echo

- BIOMARKERS
  - FIRST MARKER TO RISE : MYOGLOBIN
  - LAST MARKER TO RISE : LDH
  - AST : Liver / Skeletal muscle / Coronary specific
    ALT : Liver specific

↓

∴ ↑ AST & Ⓝ ALT ⟶ Non hepatic causes : • Rhabdomyolysis
• MI

Liver : ALT > AST Except : * Alcohol Hepatitis (∵ def Pyridox. phos)
* Cirrhosis

- CPK : – seen at : 2-4hrs
  Peak : 24hrs
  Normalise : 48-72 hrs
  – Advantage : * Almost first to rise
  * High sensitivity
  * In the setting of Reinfarction
- TROP-T : – MOST SPECIFIC
  – Begins : 4-6 hrs
  – Peak : 48 hrs
  – Normalise : 7-10 days (if Trop-I : 14 days)

TROP-I : USED IN RENAL FAILURE

- CLASSIFICATION OF MI

CLASS I : Spontaneous MI (Routine MI)

CLASS II : MI due to Non CAD Causes due to ishaemic imbalance

CLASS III : MI causing death blood samples unvailable

CLASS 4a : MI Relating PCI

CLASS 4B : MI Relating to Stent thrombosis

CLASS 5 : MI Relating to CABG

- MANAGEMENT

i) NTG (3 tab) $\xrightarrow{\text{NOT RESPON.}}$ MORPHINE
ii) Aspirin 325 mg
Ticagrelor 180mg/Clopidogrel 300mg
iii) Atorvastatin 40-80 mg
iv) Anticoagulant (LMWH / FONDAPARINOX)
v) Planning for procedure $GP_{IIB-IIIA}^{\ominus}$

C/I of NITRATES
- SEVERE HYPOTENSION
- WITH SILDENEFIL

2 MORE DRUGS TO START IN FIRST 24 HRS

vi) BETA BLOCKERS - C/I : AV BLOCK
LV FALURE
BRADYCARDIA (<60)

vii) ACE INHIBITORS

SALVAGING OF MYOCARDIUM : THROMBOLYSIS V/S PRIMARY PCI

- GOLDEN HOUR:

The benefit of intravenous thrombolysis is extremely time sensitive and when treatment is established in the First 2hr after symptom onset, the so called GOLDEN HOUR, survival dramatically decreased.

i.e THROMBOLYSIS have survival benefit if initiated within 2 hrs.

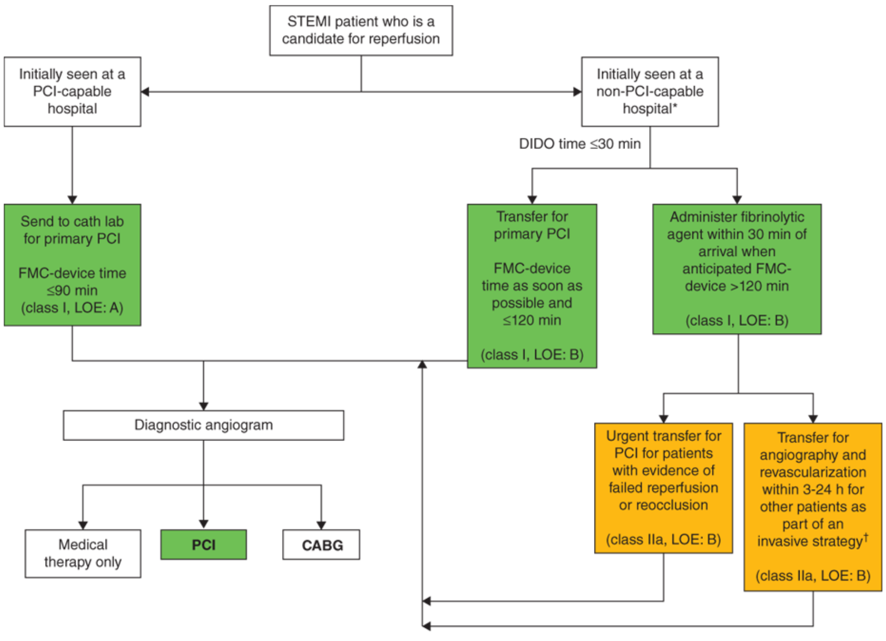
if it's within 30 mnts ⟶ AS GOOD AS PCI
if it's within 1 hr ⟶ 90% as good as PCI
if it's within 2 hr ⟶ Definite survival benefit.
Can be done up to ⟶ 12 hrs
>12hr ⟶ No benefits

- PCI is superior to Thrombolysis
- In PCI
  DOOR TO BALLOON TIME : <90 minutes
  if you are transffering : <120 minutes
- Usually Thrombolysis soon followed by PCI }
  It's associated with severe bleeding } FACILITATED PCI

- PHARMACOINVASIVE APPROACH :
  Do Thrombolysis at first possible point
  ↓
  And do PCI Later (3-24hrs Late) with full dose of HEPARIN / $GP_{IIB-IIIA}$

FACLITATED PCI	PHARMACOINVASIVE PCI
1. PCI Capable Hospital	1. Admitted to Non PCI Hospital
2. Decision for PCI taken + Pharmacological	2. PCI is back up after Lysis
3. PCI Done within 120mnts	3. PCI done after 3hrs
4. Antiplatelet I	4. Adequate antiplatelet.
5. Prothrombotic effects	5. Prevent occlusion
6. Fearing Bleeding	6. No risk of bleeding

- RESCUE PCI : Done in failed Thrombolysis. Not preffered.


STEMI patient who is a candidate for reperfusion
Initially seen at a PCI-capable hospital
Initially seen at a non-PCI-capable hospital*
DIDO time ≤30 min
Send to cath lab for primary PCI
FMC-device time ≤90 min
(class I, LOE: A)
Transfer for primary PCI
FMC-device time as soon as possible and ≤120 min
(class I, LOE: B)
Administer fibrinolytic agent within 30 min of arrival when anticipated FMC-device >120 min
(class I, LOE: B)
Diagnostic angiogram
Medical therapy only
PCI
CABG
Urgent transfer for PCI for patients with evidence of failed reperfusion or reocclusion
(class IIa, LOE: B)
Transfer for angiography and revascularization within 3-24 h for other patients as part of an invasive strategy†
(class IIa, LOE: B)

Source: Habib Samady, William F. Fearon, Alan C. Yeung, Spencer B. King III: Interventional Cardiology, 2nd Edition
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THROMBOLYSIS

- DOC : TENECTEPLASE : Advantages →
  1. FIBRIN SPCIFICITY Veryhigh
  2. FIBRIN AFFINITY High
  3. NO ALLERGIC REACTION
  4. SINGLE BOLUS ADMINISTRATION (0.5mg/kg)
  5. Patency after 1hr : >75%
  6. RISK OF BLEEDING LESS

  → ↑ Risk of ICH

- STREPTOKINASE : - 1.5 MU over 30-60min
- RETEPLASE : - 10U over 2min

  ↓ ½ hour gap

  10U over 2min
- ALTEPLASE

- C/I FOR THROMBOLYSIS

  ABSOLUTE C/I

  1. ACTIVE INTERNAL BLEEDING
  2. INTRACRANIAL NEOPLASM
  3. HEMMORRHAGIC CVA at any time
  4. CVA/TIA within one year
  5. Suspected Aortic dissection

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ECG CHANGES IN ACUTE CORONARY SYNDROME

SURFACES & BORDERS OF HEART

SURFACES

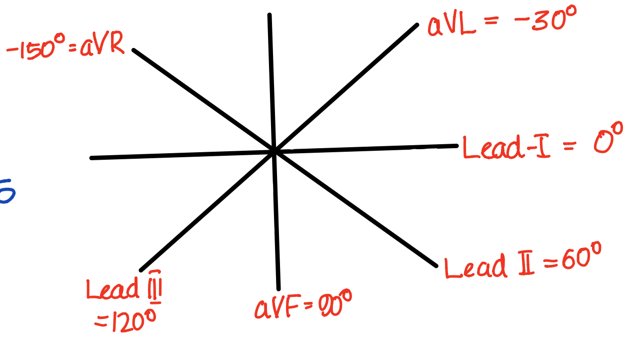
1. Anterior / Sternocostal : RV
2. Posterior : LA
3. Inferior / Diaphragmatic : RV+LV

BORDERS

1. Right border : RA
2. Left border : LV+LA
3. Superior border : LA+RA+ Aorta + Pulmonary trunk
4. Inferior border : RV+ LV

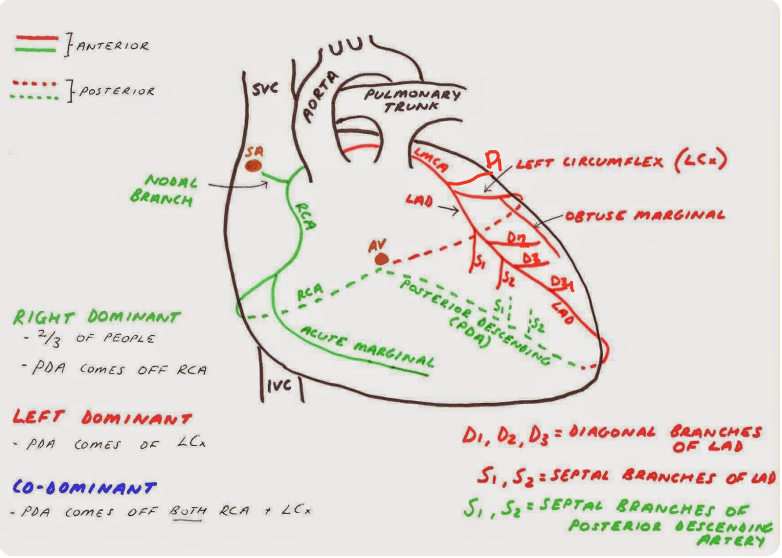
LEADS

1. INFERIOR WALL : II , III , aVF
2. ANTERIOR WALL : V_2, V_3, V_4
3. SEPTUM : V_1, V_2
4. APEX : V_4 (Predominantly), V_5
5. RV FREE WALL : V_{4r}, V_{5r}, V_{6r}
6. POSTERIOR WALL : V_7, V_8, V_9
7. LATERAL LEADS : V_5, V_6
8. HIGHLATERAL LEADS : I , aVL



CIRCULATION

- RCA - in 45-50% : MC INVOLVED VESSEL
- LCA - in 36-40%
- LCX - in 10-15%



Right Coronary artery

- Start in the Right AV Sulcus

↓

Supplies SA Node

a) ACUTE MARGINAL ARTERY
Supplies the RV Free wall

b) Goes posteriorly & supply

Posterior Wall + Branch to AV Node

c) Posterior descending Artery : SUPPLY INERIOR WALL OF HEART

✓ RV FREE WALL IS SUPPLIED ONLY BY : RCA

✓ POSTERIOR WALL → RCA/LCX or RCA & LCX
INFERIOR WALL → RCA or LCX
} IN 85% RCA
15% LCX

✓ If the posterior descending artery is originating from
- → RCA : Right Dominant (in 85%)
- → LCX : Left Dominant (in 15%)

↓ Supply inferior wall

✓ If the patient have one proximal wall occlusion } RVMI + IWMI + PWMI (Post. Wall MI)

If the level of occlussion after marginal artery } IWMI + PWMI

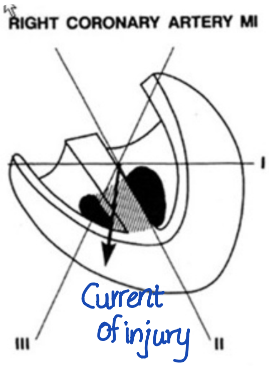
If the occlusion is at PDA - Only IWMI (Inf. wall MI)

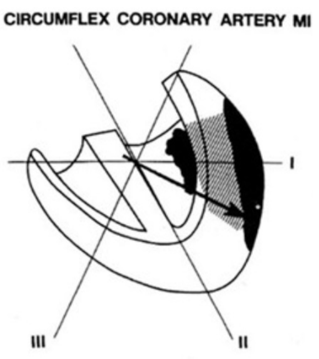
✓ COMPLICATION :
a) Heart block : as it supply SA & AV Nodes
b) Atrial fibrillation
c) Pappillary muscle rupture ⟶ Acute MR
d) RVMI

NOTE :
Vagal afferent fibers can produce BRADYCARDIA by Reflux stimulation
So in an Inferior wall MI, Bradycardia may be due to
(i) Vessel occlusion
(ii) Reflux stimulation of vagal afferent
But in Anterior wall MI, Bradycardia denotes
EXTENT OF ISCHAEMIA

LEFT CIRCUMFLEX ARTERY

- Originate from Left Main Coronary artery (LMCA)
- Supplies
  - (i) Lateral wall of heart through OBTUSE MARGINAL ARTERY
  - (ii) Posterior wall of heart
  - (iii) 10-15% cases Inferior wall of heart
- NOTE : NO RVMI Here.


RIGHT CORONARY ARTERY MI


CIRCUMFLEX CORONARY ARTERY MI

NOTE:

RCA Occlusion	LCX Occlusion
↓	↓
ST elevation in Lead III > II	ST elevation in Lead II > III

- LOOK FOR RECIPROCAL CHANGE
  Here every lead got reciprocal change
  i.e it's ST ELEVATION
- ST Elevation in Lead II, III, aVF

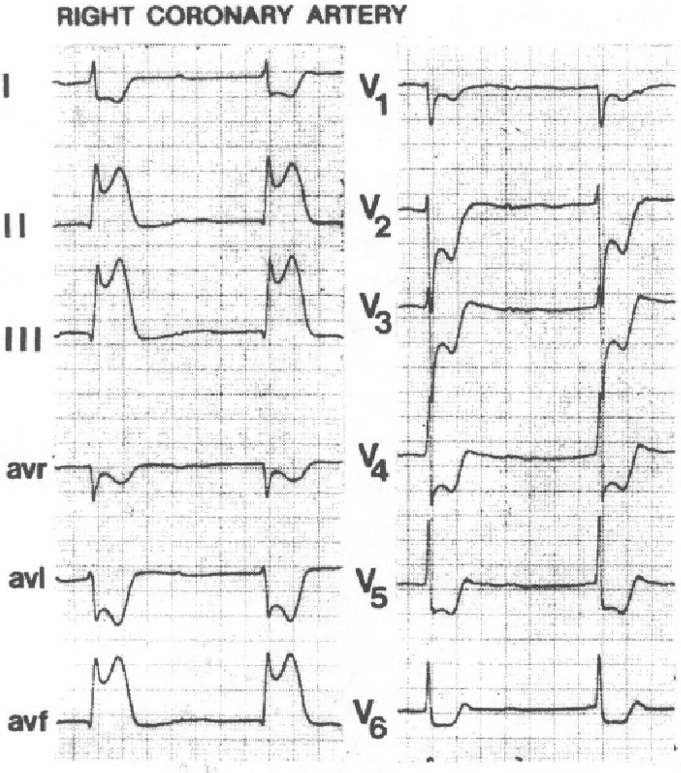
↓

Inferior Wall MI

RCA (in 85%) LCX (15%)

Here ST elevation is more in Lead III > II
∴ it's due to RCA

- As it's by RCA, Check for
  a) RVMI
  On a 12 Lead ECG look for V_1-V_2
  In RVMI V_1 will show DISORDANCE with V_2
  ∴ Here **RVMI** also
  b) PWMI
  On a 12 Lead EGG look for V_2
  In PWMI V_2 show ST Depression & Reciprocal changes of ST Elevation
  ∴ Here **PWMI**
- Now find the Rate: Here bradycardia
  ∴ SA node may also effected
- As it's IWMI + PWMI + RVMI **Level of occlusion very proximal**


RIGHT CORONARY ARTERY

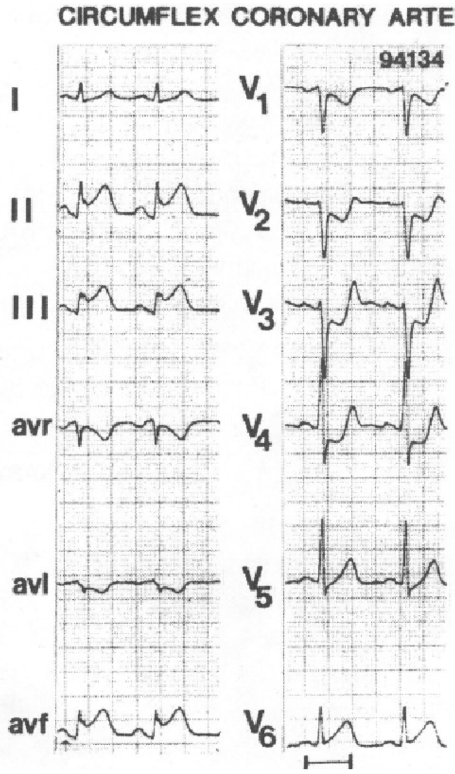
NOTE:

To find RVMI
(i) ST Elevation in $V_{3r}, V_{4r}, V_{5r}, V_{6r}$
(ii) In 12 lead ECG
V_1 will show Discordance with V_2

Usually V_1 minimal ST depression / flat
(∵ injury vector will pull up the Right side lead V_1 ∴ minimal depression)

NOTE:

To find out PWMI
(i) On V_7, V_8, V_9
(ii) On 12 lead ECG look for V_2
will show ST depression & Reciprocal change of ST Elevation



- ST Elevation : Lead II, III & aVF

↓

∴ IWMI

- ST elevation in lead II & III

↓

∴ LCX effected

- ∴ check for a) PWMI

V_2 show ST depression

↓

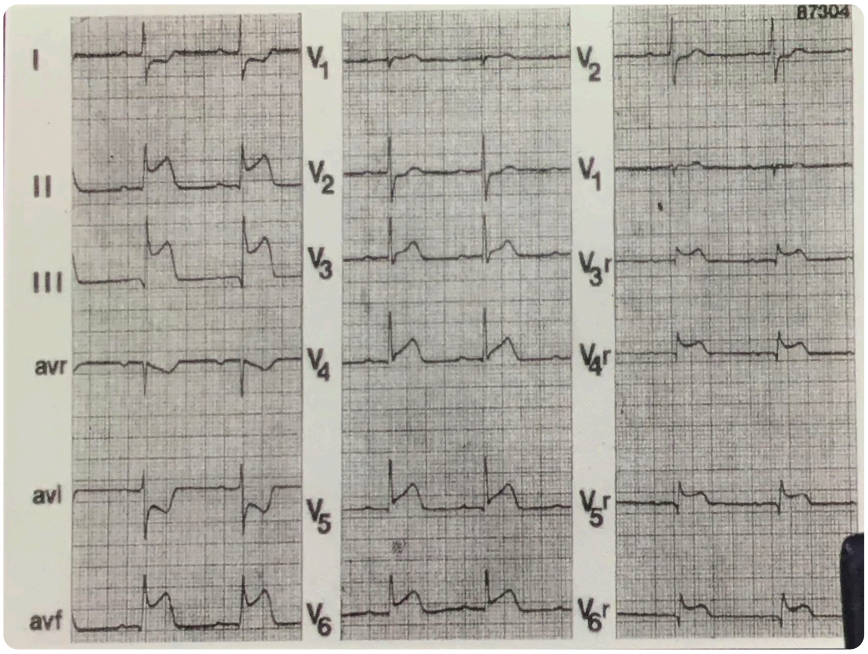
∴ it's PWMI also

b) LWMI

Check for V_5, V_6 ST elevation

∴ Here LWMI also

∴ IWMI + PWMI + LWMI



- ST ELEVATION IN II, III, aVF
- RECIPROCAL CHANGES in I & aVL
- ST ELEVATION III > II

↓

∴ IWMI with RCA occlusion

- V_1 showing ST Elevation usually it should be depressed.

∴ RVMI

(Also ST elevation in V_{3r}, V_{4r}, V_{5r}, V_{6r})

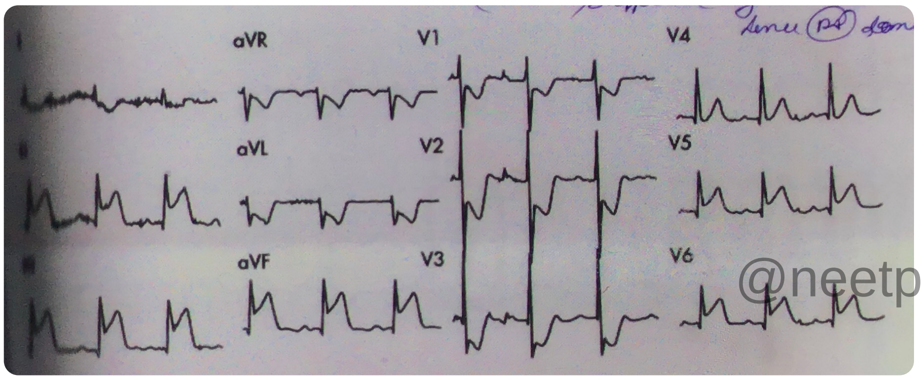
- V_2 showing ST Elevation.

∴ Posterior wall is spared

- ∴ IWMI + RVMI

But here V_5, V_6 show ST elevation

The RCA in this case supply the Lateral wall also. This is called WRAP AROUND RCA

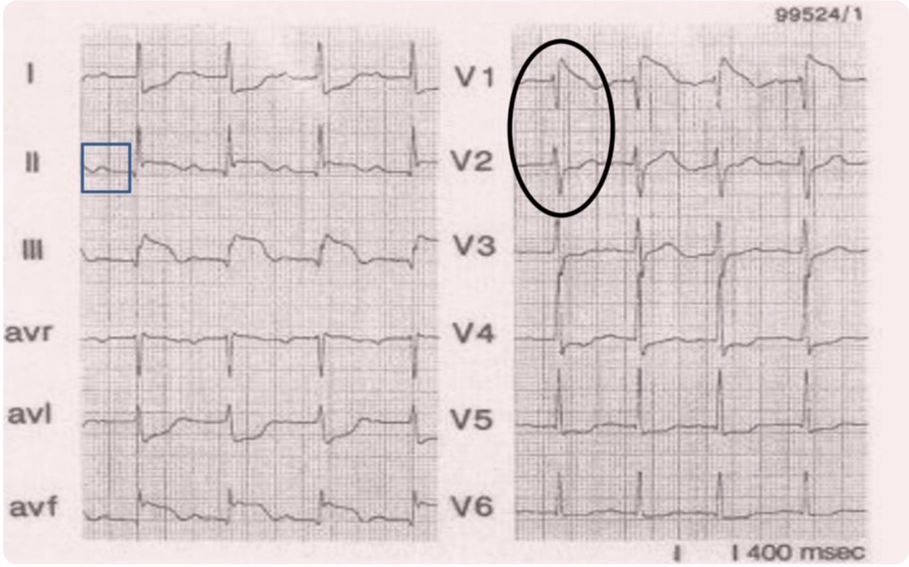


- Lead III > II ∴ RCA

(∵ aVL show max depression)

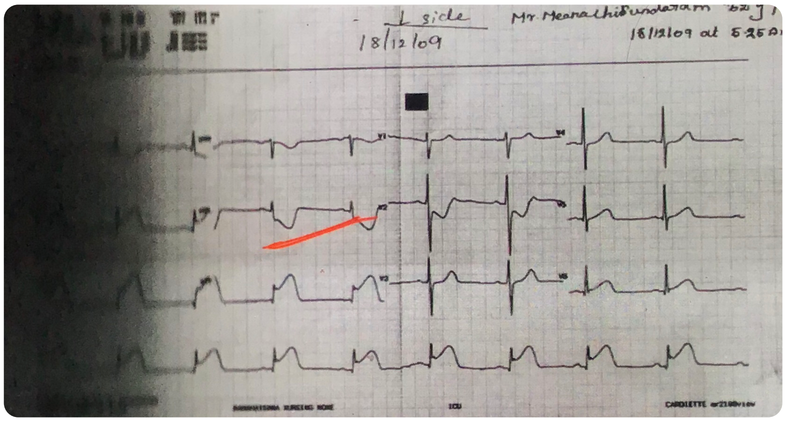
- V_1-V_2 discordance ∴ ∴ RVMI
- V_2 ST depression ∴ ∴ PWMI
- V_5 & V_6 ST elevation ∴ ∴ Lateral wall is also supplied

∴ Wrap around RCA

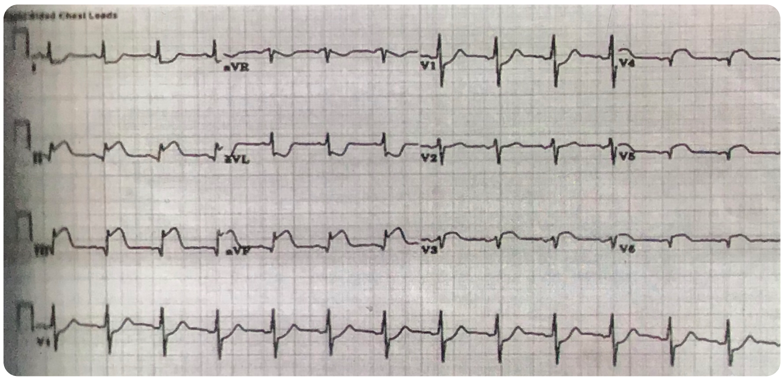


- Lead III > II : ∴ LCX
- Reciprocal changes in aVL
- V_1 showing ST elevation
  - → ∴ RVMI
- Lead II, III, aVF ST elevation
  - → IWMI
- No depression of ST : ∴ No PWMI
- Rate ≙ 100 beats/min.

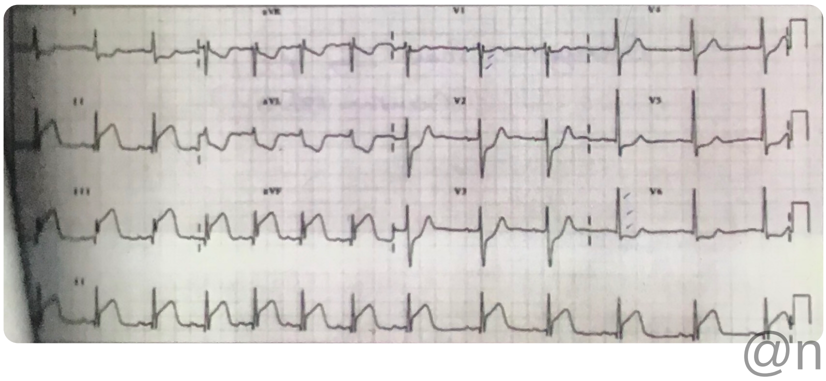
∴ RVMI + IWMI



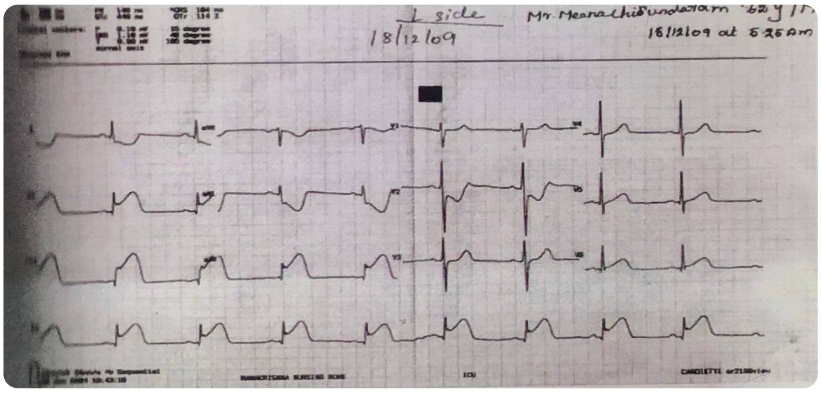
- Lead III > II → ∴ RCA
- V_1 discordance with V_2
  - → ∴ RVMI
- V_2 depression of ST
  - → ∴ PWMI
- ∴ it's IWMI + RVMI + PWMI proximal occlussion



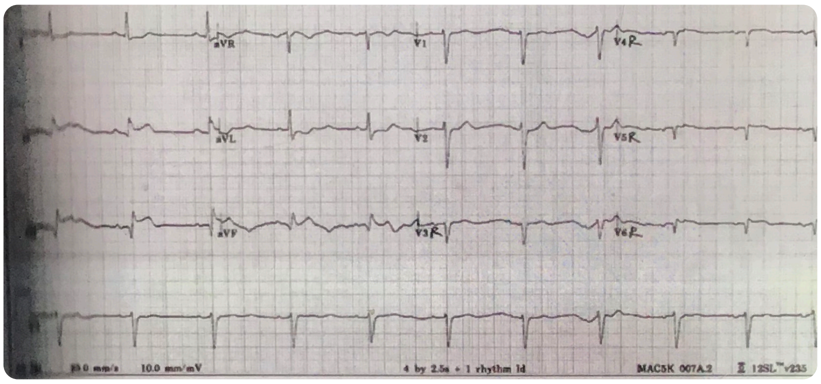
- Lead III > II : RCA Occlusion
- No depression in V_2
  - → No PWMI
- V_1 & V_2 showing some discordance
  - → No confirmation
- ∴ only IWMI / Distal RCA



- Lead III > Lead II : RCA
- Discordance of V_1 & V_2
- V_2 ST depression : ∴ PWMI
- ∴ IWMI + RVMI + PWMI

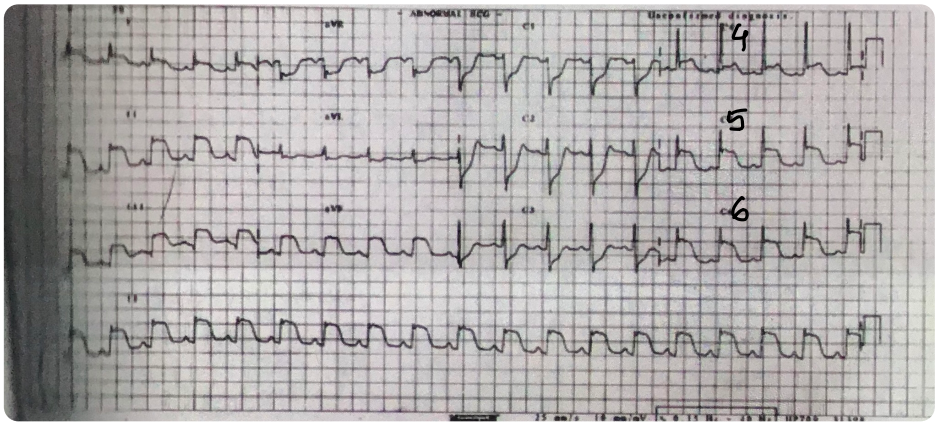


- Lead III > Lead II : RCA
- V_1-V_3 discordance
- V_2 depressed
- ∴ IWMI + PWMI + RVMI

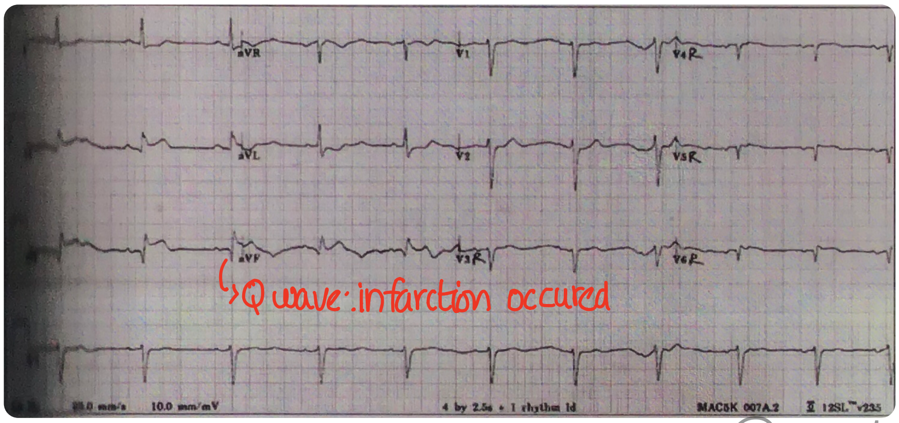


- Lead III > II ∴ RCA
- V_1 ST flat :
  V_{4r}, V_{5r}, V_{6r} : Mild degree of ST elevation (RVMI)
- V_2 ST no depression : ∴ No PWMI

∴ IWMI + RVMI
(∴ Proximal RCA occlu.)

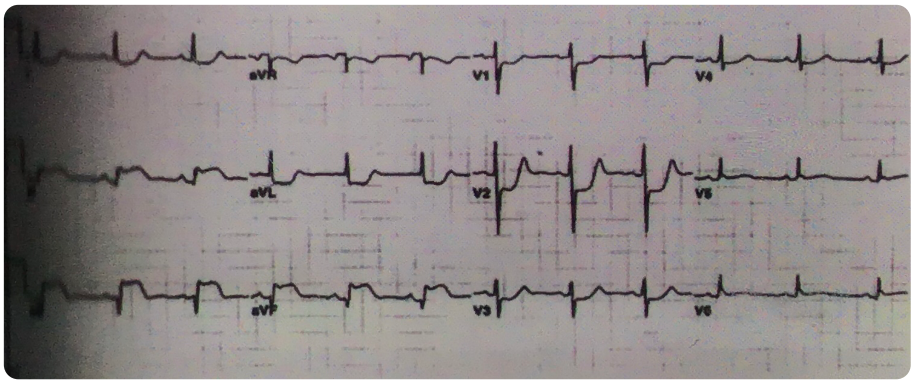


- Lead II > III : LCX
- V_5, V_6 ST elevation
- V_2 ST depression
- ∴ PWMI + LWMI + IWMI

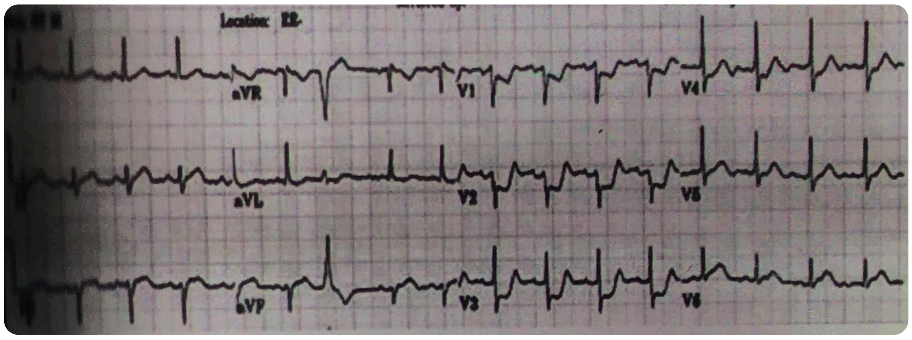


- Lead III > II with a Q wave
  ↳ ∴ RCA
- No ST depression on V_2 : No PWMI
- V_1 flat :
  V_{4r}, V_{5r}, V_{6r} : ST elevation

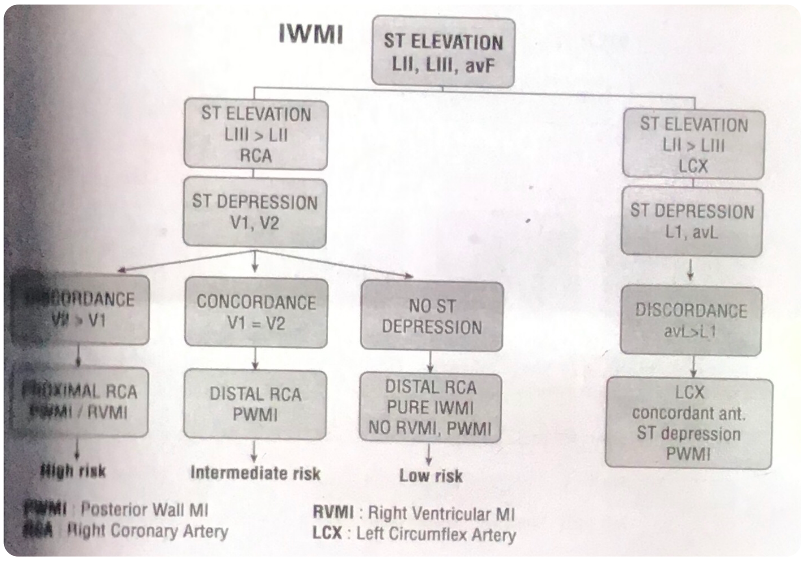
∴ RVMI
∴ Proximal RCA



- Lead III > II : RCA
- V_1 - V_2 Discordance
  ↳ ∴ RVMI
- V_2 depression : ∴ PWMI
- ∴ IWMI + PWMI + RVMI



- ST depression in V_1, V_2, V_3
  2 possibilities
  - Antero Septal NSTEMI
  - PWMI
- Profound depression in V_2
  Chance of PWMI is high
  (V_1 depression < V_2)
  ∴ Do a BEDSIDE ECHO



PWMI : Posterior Wall MI
RCA : Right Coronary Artery
RVMI : Right Ventricular MI
LCX : Left Circumflex Artery

Summary

IWMI ⟶ Reciprocal changes in I, aVL (or) V_1 - V_6

Lead III > II ST Elevation
↓
RCA
- V_1-V_2 Discordance with ST depression
  RVMI(+)
  PWMI(+)
- V_1, V_2 Depression with no disordance
  PWMI(+)
- V_1, V_2 Normal
  IWMI

Lead II > III ST Elevation
↓
LCX
↓
LWMI : ST elevation V_5, V_6
PWMI : ST depression in V_1, V_2, V_3
↓
Predominant

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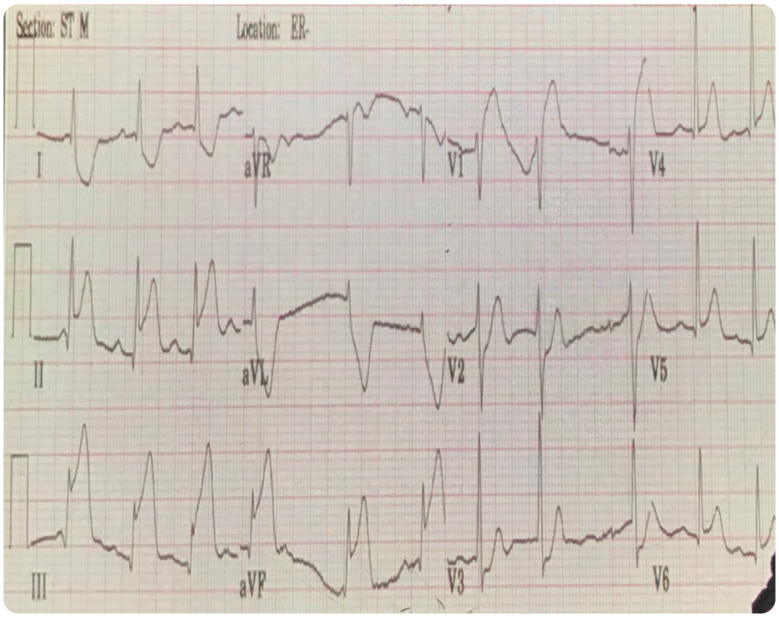
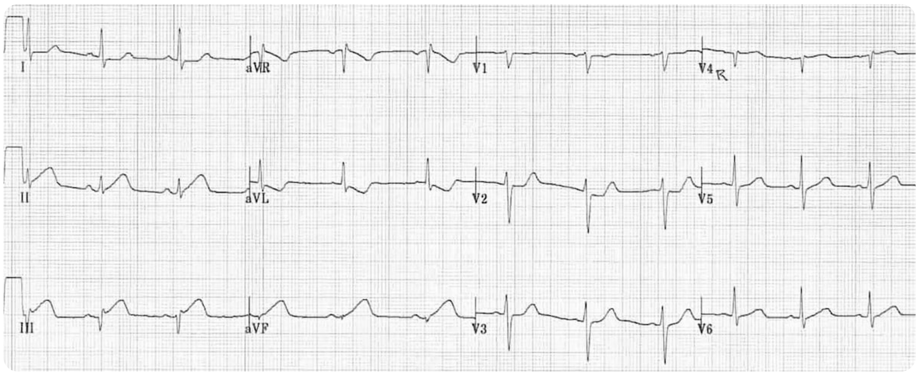
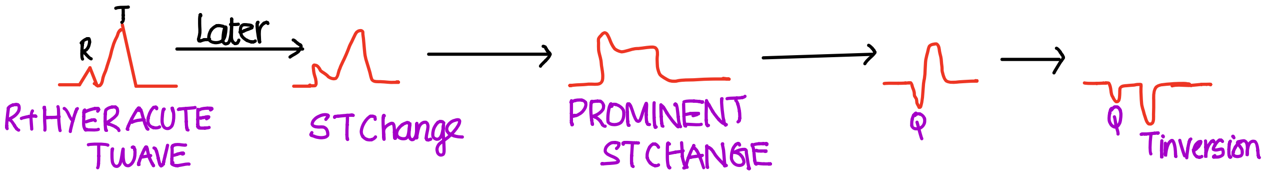


- Lead III > II : RCA
- V_1 showing ST elevation : ∴ RVMI
- Some depression in V_2 : ∴ PWMI
- But here
  PR interval are not regular
  PP interval are regular
  ↓
  3° Heart block
- IWMI + Complete Heart block
  Pt require Pacing.

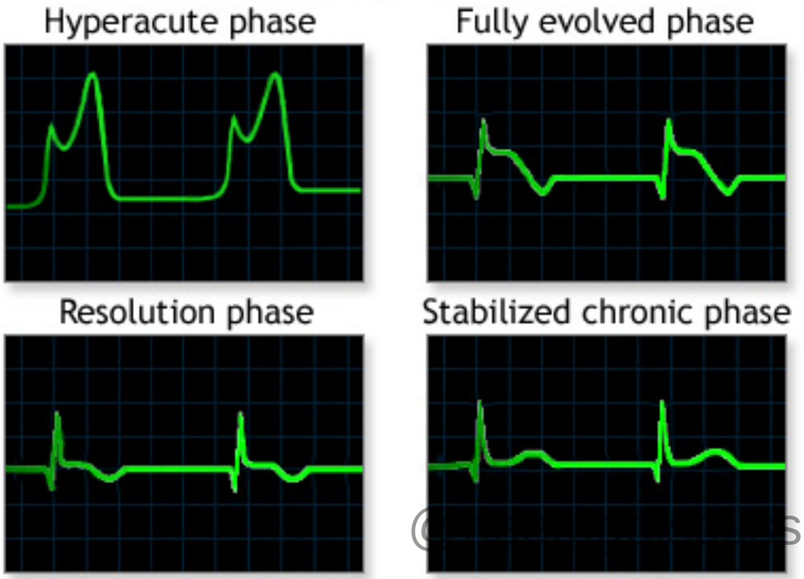


- LEAD III > II
- V_1-V_2 DISCORDANCE
- V_{4R} : ST elevation
- ∴ RVMI + IWMI

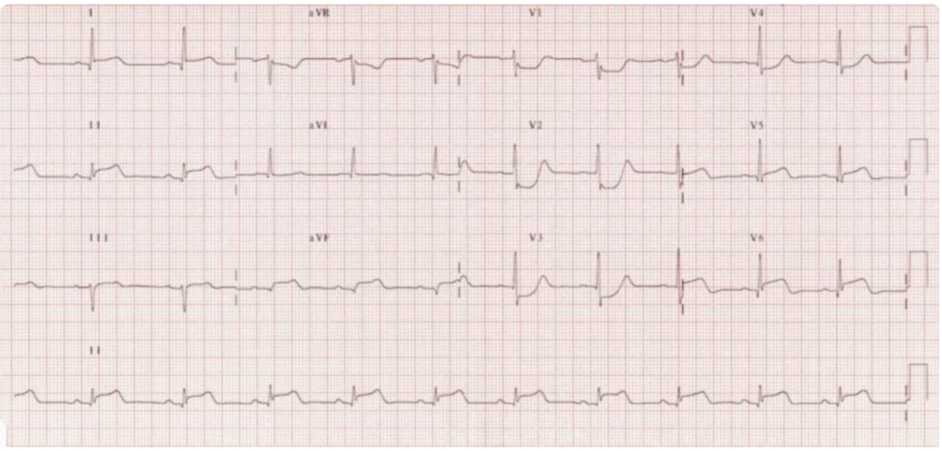
PROGRESSION OF MI



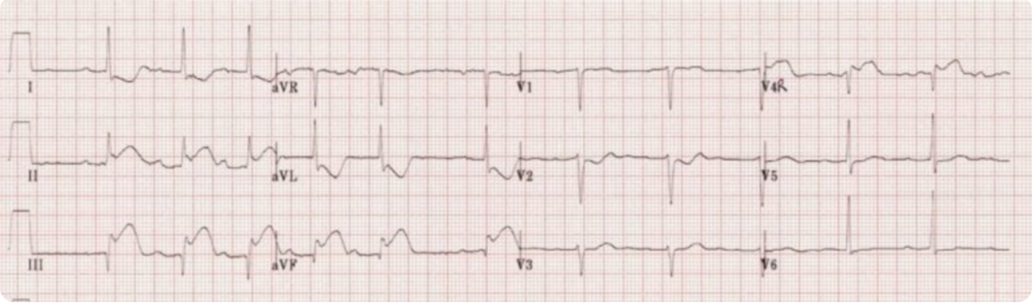
Post Myocardial Infarction



@ sion
More than just notes...



- LEAD II > III : LCX
- V2 DEPRESSION OF ST
  ↓
  PWMI
- V5, V6 ST Elevation
  ↓
  LWMI



- LEAD III > II : RCA
- V1 – V2 DISCORDANCE
- RVMI, IWMI, Most often PWMI.

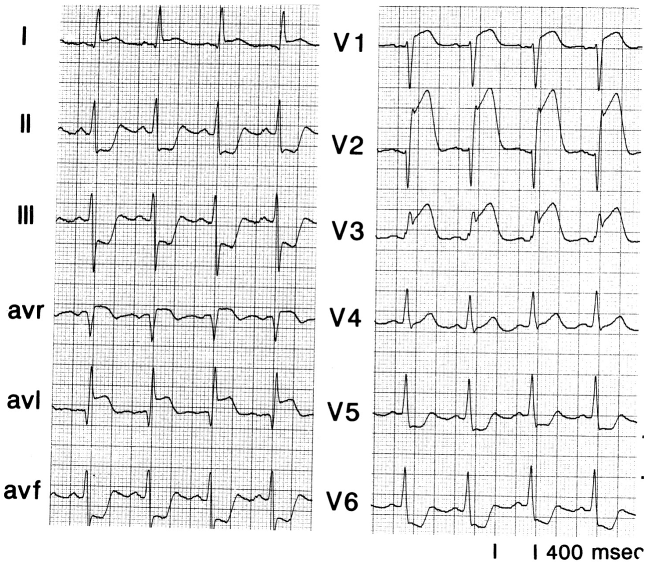
LEFT CORONARY ARTERY

- Originate from Left interventricular sulcus
- First Branch : DIAGONAL BRANCH (D1) : Parrelel To CIRCUMFLEX A.
  → Supply BASAL ANTEROLATERAL WALL is also called as High Lateral
  → Leads I, aVL
- SEPTAL BRANCH (S1) : Supply
  ANTERIOR 2/3rd of INTER VENTRICULAR SEPTUM
  CONDUCTING SYSTEM DISTAL TO BUNDLE of HIS
  Leads V1 – V2
- DIAGONAL BRANCH (D2) : Supply
  ANTERIOR WALL + APEX
  Leads V2 – V4
- LEADS V5 – V6 : BY LCX or LAD

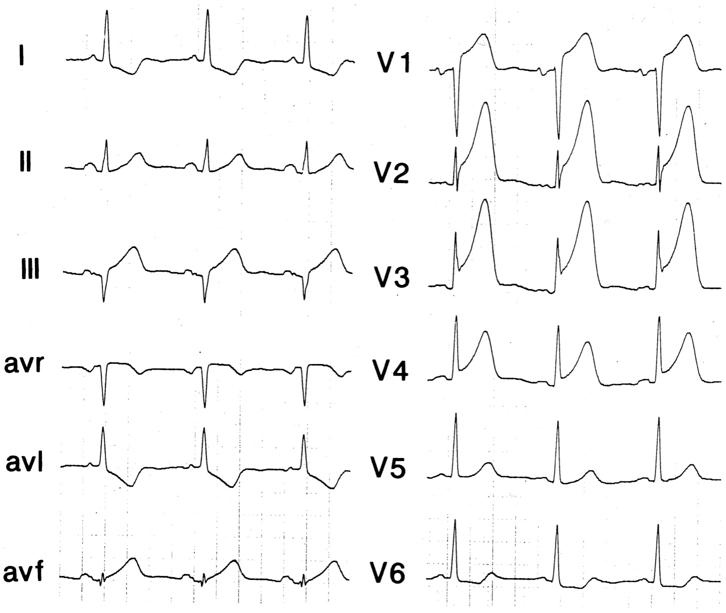
Occlusion Proximal LAD	Occlusion afte origin of LCX	V1 - V6 involvment
↓ involve V1-V6, I, aVL	V1-V4, I, aVL	ANTEROLATERAL MI
EXTENSIVE ANTERO-LATERAL MI	ANTEROSEPTAL MI	V1 - V2 involvement
		SEPTAL MI

✓ Anterior wall MI is more severe as it can cause
- (i) TACHYARRYTHMIA
- (ii) RBBB > LBBB
- (iii) CARDIOGENIC SHOCK

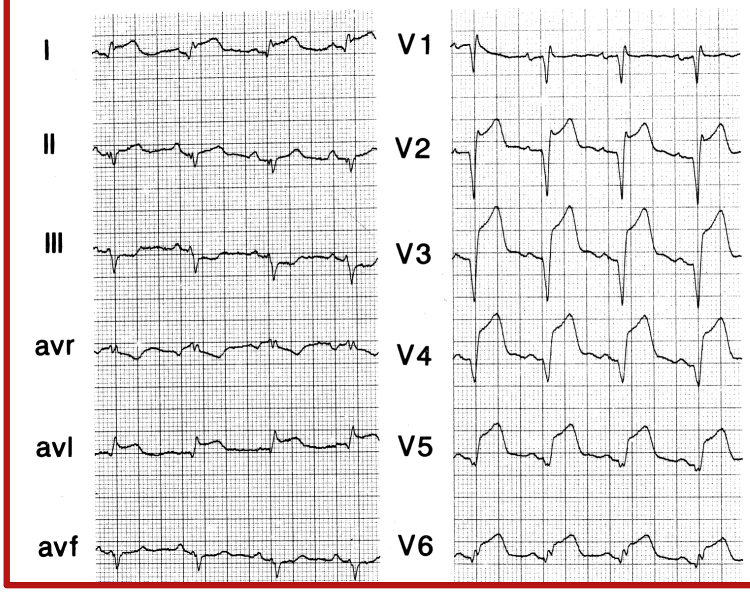
✓ SITE OF LESION
- (i) PROXIMAL (before D_1 & S) ⟶ EXTENSIVE ANTEROLATERAL MI
- (ii) Between D_1 & S_1 ⟶ ANTEROSEPTAL MI
- (iii) DISTAL TO S_1 ⟶ PURE ANTERIOR WALL MI



- RECIPROCAL CHANGES ARE SEEN
- ST ELEVATIONS : V_1, V_2, V_3, I, aVL ↓ Significant ⟶ ∴ Diagonal br involved ↓ SITE OF LESION ABOVE THE ORIGIN OF DIAGONAL BRANCH
- V_1, V_2 involved ↓ ∴ SEPTUM involved
- V_2, V_3 ST ELEVATION : ANTERIOR WALL
- ∴ LAD OCCLUSION PROXIOMAL TO D_1
- NO ST ELEVATION ON V_5, V_6 ∵ LCX Supply



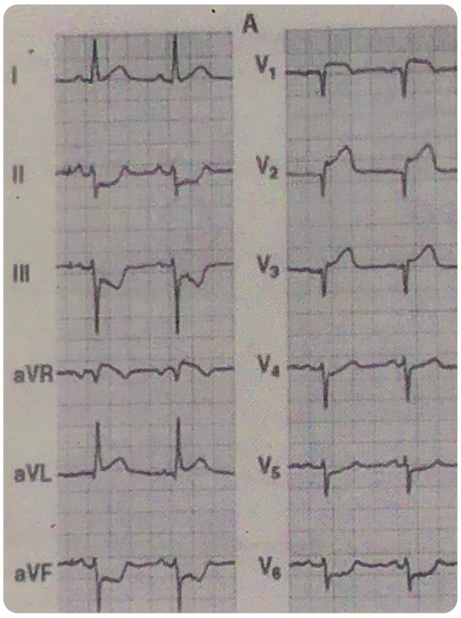
- RECIPROCAL CHANGES ARE SEEN
- V_1 – V_4 ST ELEVATION: ANTERO SEPTAL
- I & aVL : NO ST ELEVATION
  : ∴ D_1 SPARED
- V_5 & V_6 : NO ST ELEVATION
  : ∴ CIRCUMFLEX SPARED
- V_1 & V_2 : ST ELEVATION
  : ∴ BEFORE SEPTAL ARTERY
- ∴ SITE OF OCCLUSION : BETWEEN D_1 & S_1



- RECIPROCAL CHANGES ARE SEEN
- I, aVL : ST ELEVATION
  : ∴ LESION PROXIMAL TO FIRST DIAGONAL (or) LCX OCCLUDED
- V_5 - V_6 : ST ELEVATION
- V_1 - V_6 : ST ELEVATION

↓

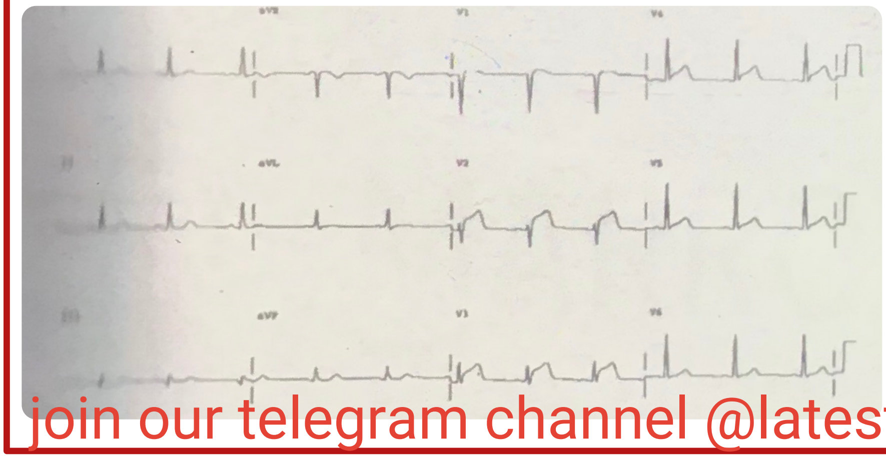
∴ VERY PROXIMAL LCA OCCLUSION



- RECIPROCAL CHANGES ARE SEEN
- I, aVL : ST ELEVATION
- V_1 - V_2 : ST ELEVATION
- V_5 - V_6 : NO ST ELEVATION (LCX SPARED)

↓

∴ PROXIMAL OCCLUSION BEFORE D_1



- NO RECIPROCAL CHANGES

↓

∴ NOT MI

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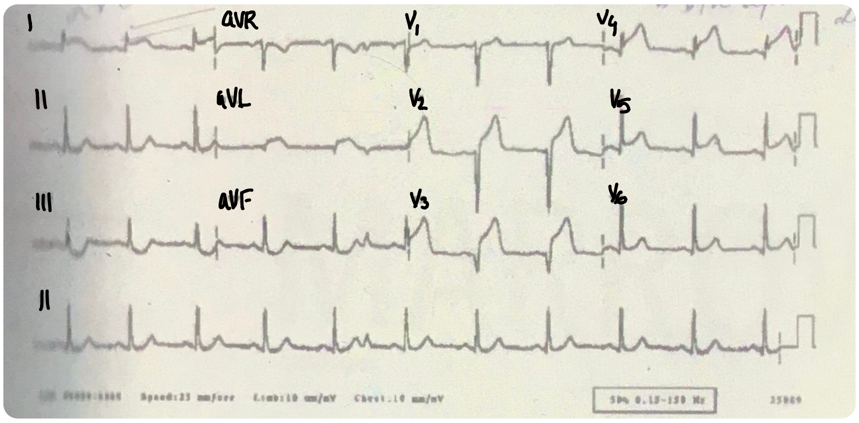
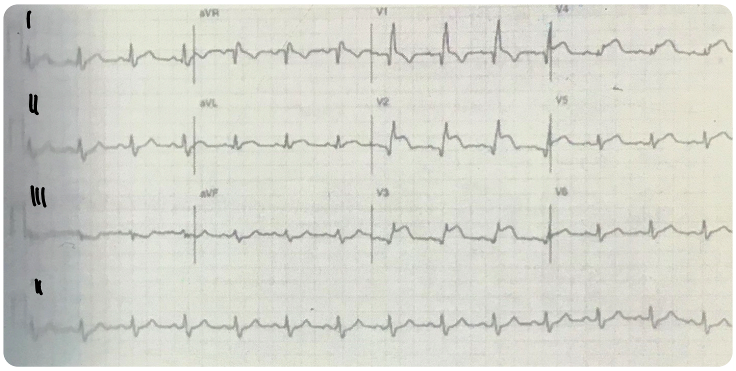
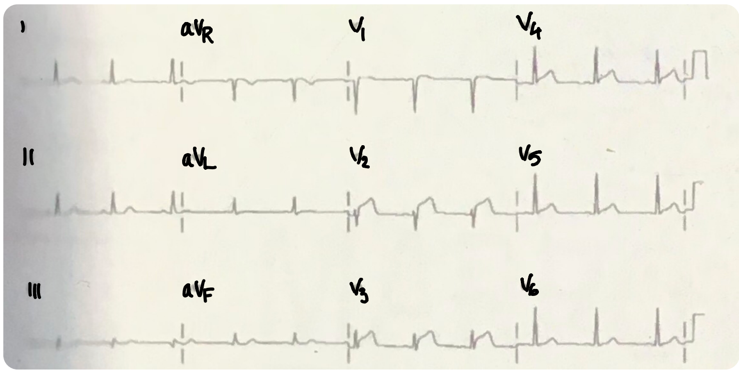


- RECIPROCAL CHANGES ARE SEEN (III & aVF)
- I & aVL : ST ELEVATION
- V_1-V_5 : ST ELEVATION
  V_6: ? : ANTEROLATERAL INVOL.
- LESION: PROXIMAL LAD OCCLUSION ABOVE D_1



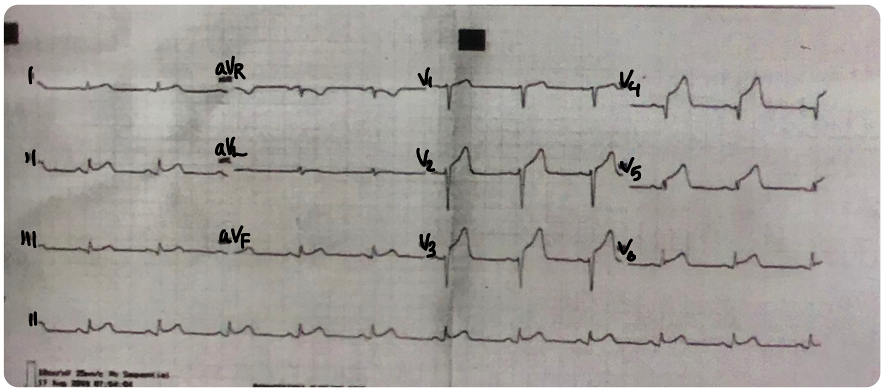
- RECIPROCAL CHANGES ARE SEEN
- I & aVL : ST ELEVATION
- V_1-V_6 : ST ELEVATION
- LESION: PROXIMAL LAD OCCLUSION ABOVE D_1



- NO RECIPROCAL CHANGES

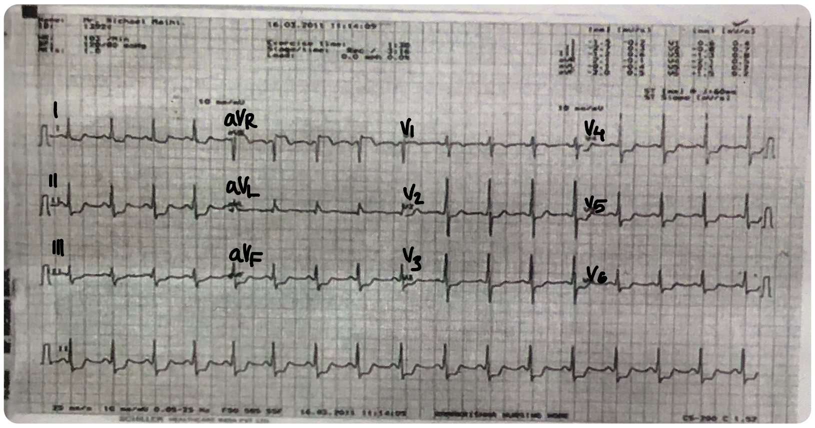
↓

∴ NOT MI



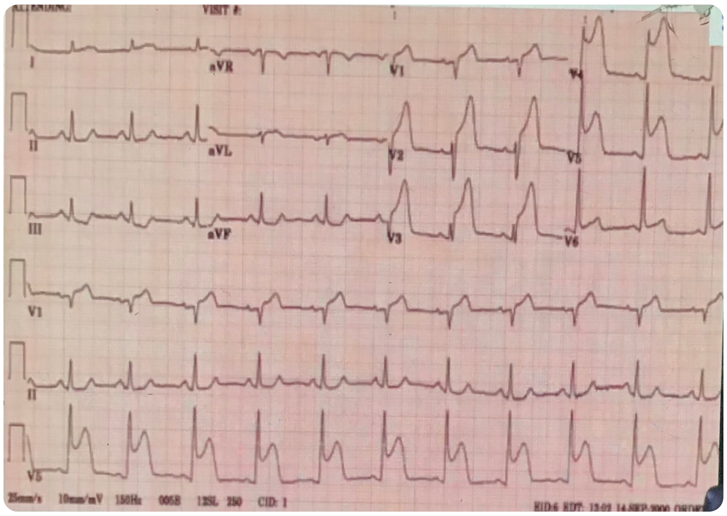
- RECIPROCAL CHANGES ARE SEEN
- V_1-V_6 : ST ELEVATION
- II - III : ST ELEVATION

→ After supplying Ant. & Lat. wall a small wrap supply INFERIOR WALL ALSO
WRAP AROUND LCA



OSTIAL LAD OCCLUSION / LEFT MAIN OCCLUSION

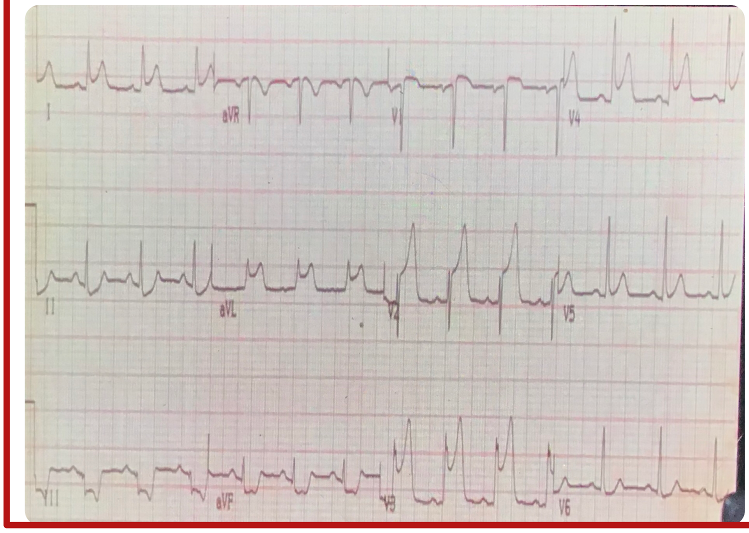
- ST ELEVATION IN aVR
- NO ROLE OF THROMBOLYSIS
  IMMEDIATELY SHIFT TO CATH LAB



- RECIPROCAL CHANGES ARE SEEN
- ST ELEVATION : V_1-V_6, I, aVL

↓

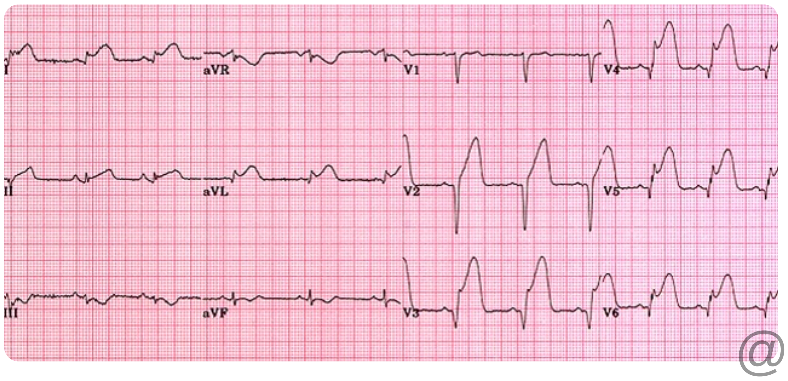
PROXIMAL OCCLUSION ABOVE THE ORIGIN OF LCX



- RECIPROCAL CHANGES ARE SEEN
- ST ELEVATION : V_1-V_4, I, aVL

↓

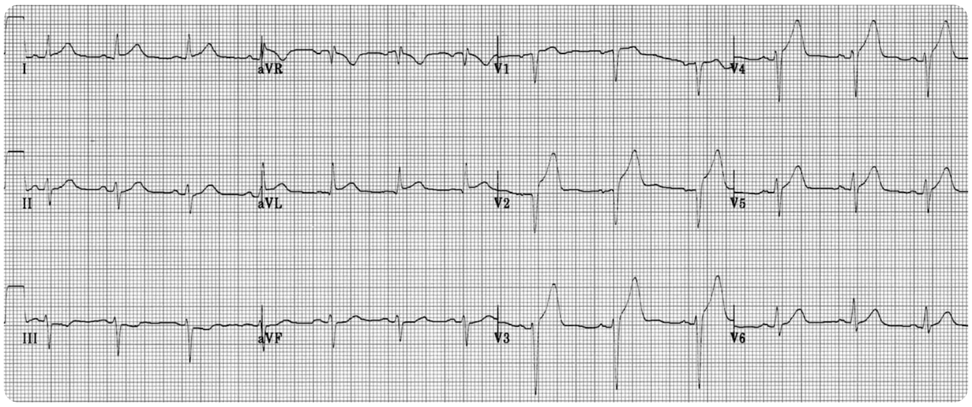
LAD above origin of D_1



- RECIPROCAL CHANGES ARE SEEN
- ST ELEVATION : V_2-V_6, I, aVL

↓

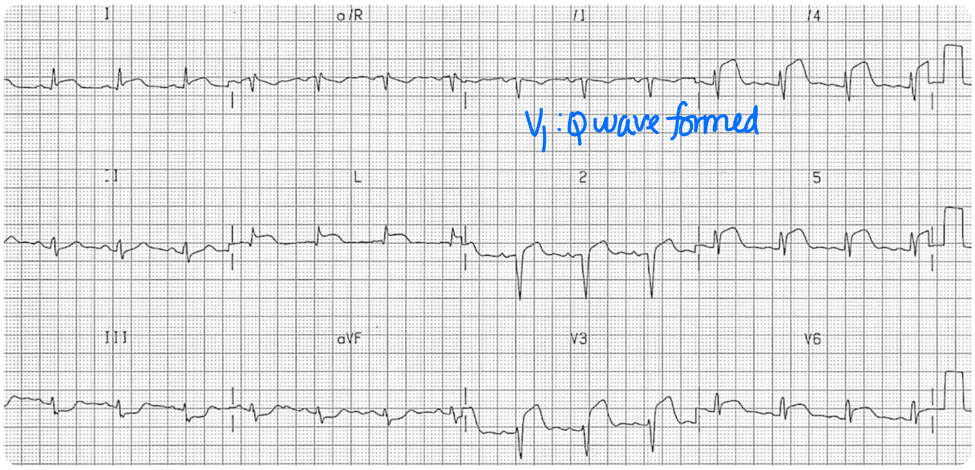
PROXIMAL OCCLUSION ABOVE THE ORIGIN OF LCX



- RECIPROCAL CHANGES ARE SEEN
- ST ELEVATION: V_1-V_4, I, aVL

↓

LAD above origin of D_1

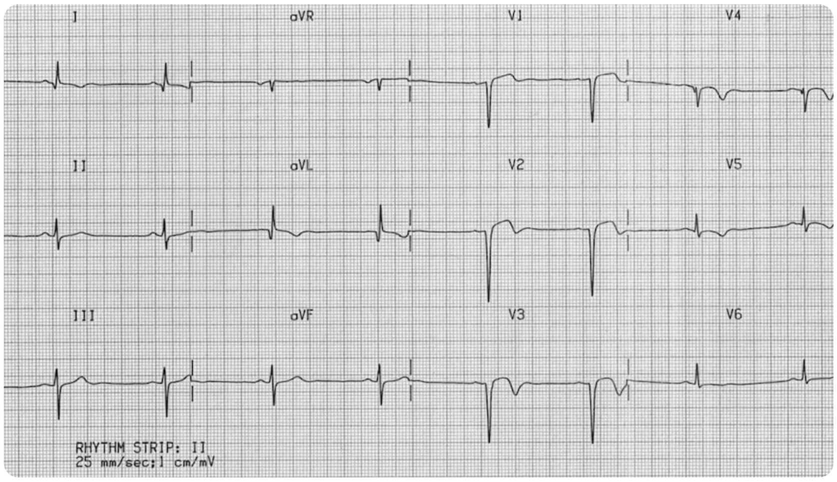


V_1 : Q wave formed

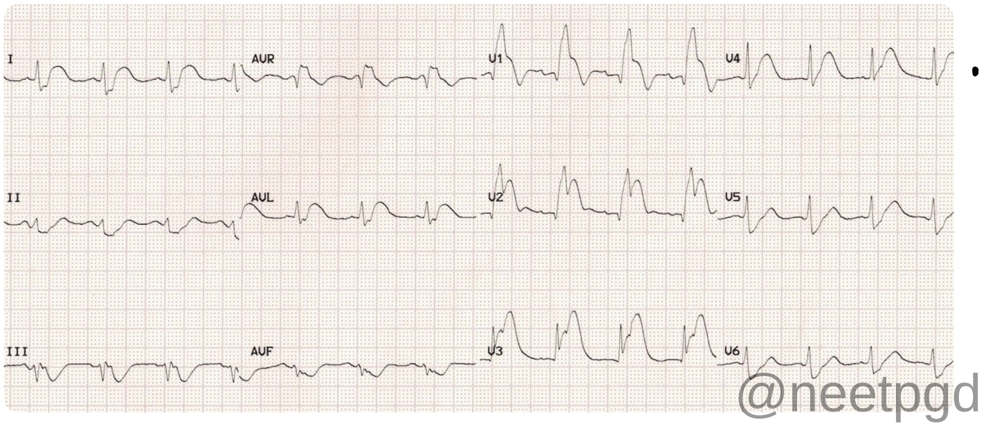
- RECIPROCAL CHANGES ARE SEEN
- ST ELEVATION: V_1-V_6, I, aVL

↓

PROXIMAL OCCLUSION ABOVE THE ORIGIN OF LCX



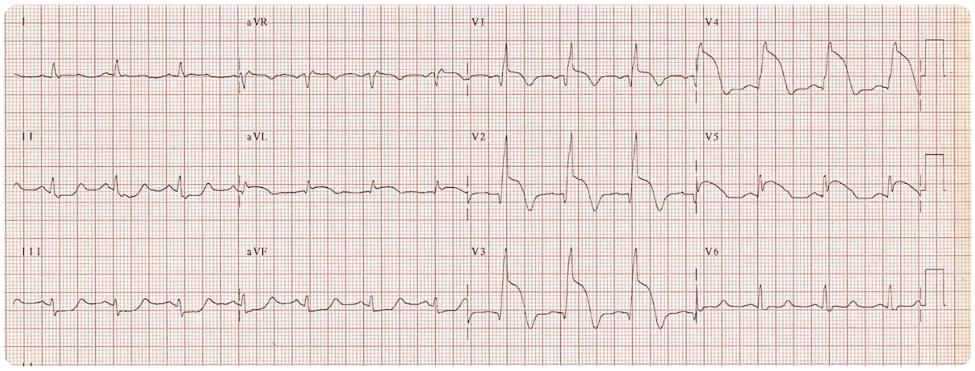
- RECIPROCAL CHANGES ARE SEEN
- NO ACTIVE ACUTE RECIPROCAL CHANGES
- QS Complex in V_1-V_2
- RESIDUAL ST ELEVATION IN V1-V3: LEFT VENTRICUR ANEURYSM MORPHOLOGY
- OLD MI WITH PERSISTENT ST ELEVATION



- ST ELEVATION IN aVR

↓

OSTIAL LAD OCCLUSION



- RECIPROCAL CHANGES ARE SEEN
- ST ELEVATION : V_1-V_6, I, aVL

↓

PROXIMAL OCCLUSION ABOVE THE ORIGIN OF LCX

SUMMARY

aVR : ST ELEVATION ⟶ Proximal ostial stenosis

I, aVL, V_5, V_6 ST ELEVATION : ABOVE CIRCUMFLEX

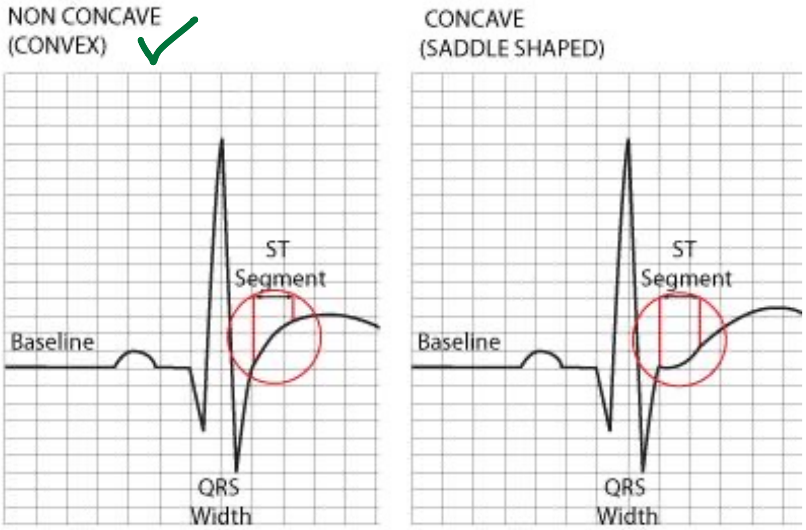
I, aVL ST ELEVATION : Proximal to D_1

V_1-V_4 ST ELEVATION : B/W D_1 & S_1 → ANTEROSEPTAL

V_2-V_4 ST ELEVATION : AFTER S_1 → PURE ANTERIOR

SIGNIFICANT ST ELEVATION

CONVEX UPWARD elevation is taken as Significant ST elevation.



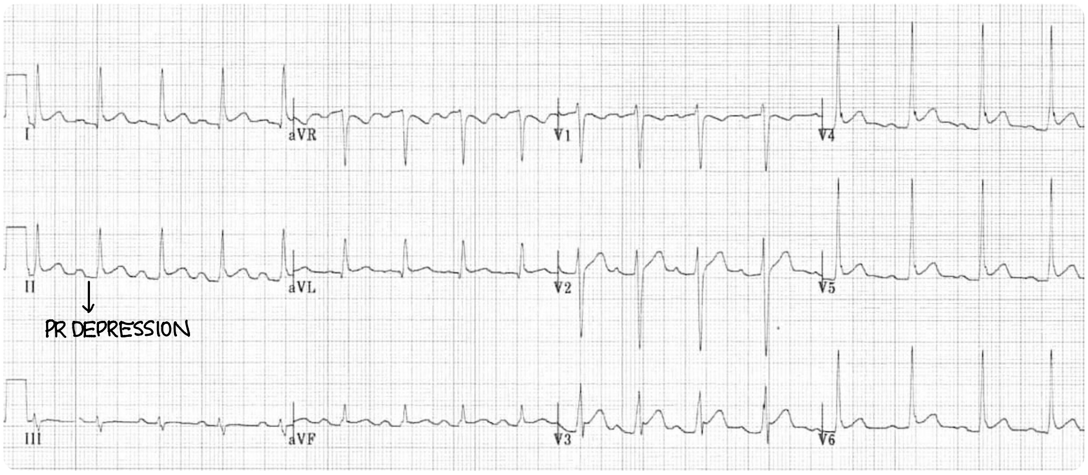
Learning Bite

A convex ST segment shape is more likely to be associated with AMI than a concave shape

Pitfall

However,do not assume that because ST segment elevation is not convex that it cannot be a STEMI

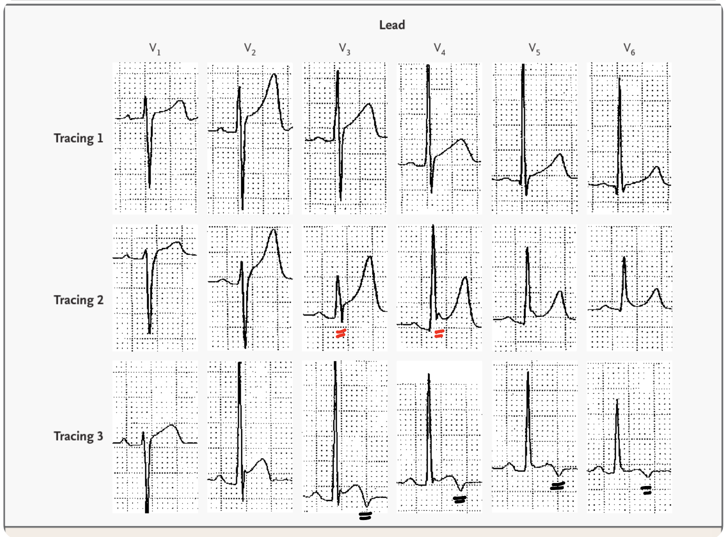
DIFFERENTIAL DIAGNOSIS OF ST ELEVATION



Here

- * LOW VOLTAGE COMPLEXES
- * GLOBAL ST ELEVATION
- * NO RECIPROCAL CHANGES
- * CONCAVE UPWARDS
- * PR SEGMENT DEPPRESSION

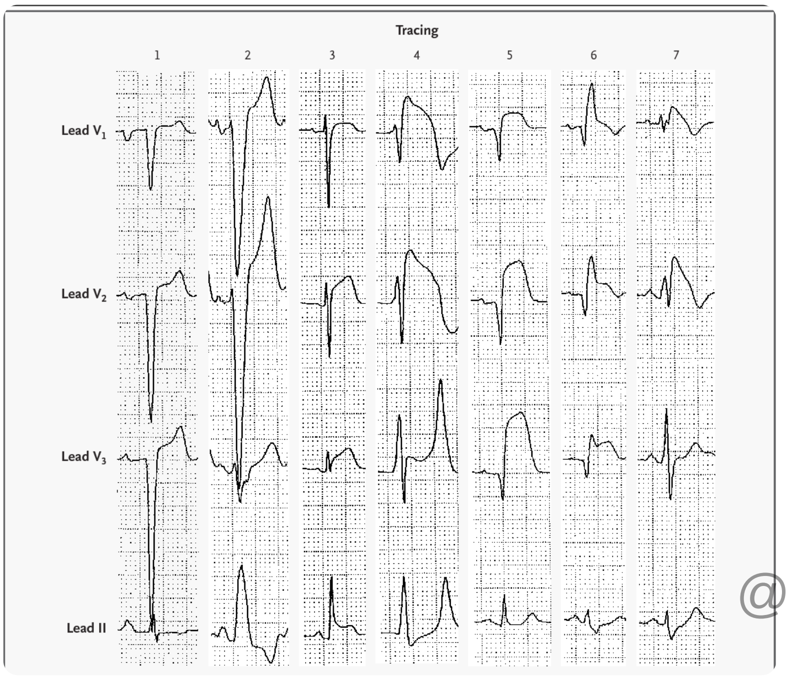
} PERICARDITIS



Tracing I: CONCAVE UPWARD ST ELE
NO RECIPROCAL CHANGES
It's NORMAL ECG

Tracing I: CONCAVE UPWARD ST ELE
NOTCH AT "J" POINT
EARLY REPOLARISATION SYN.

Tracing III: ASSYMETRICAL T-INVERSION
NORMAL VARIANT



Tracing 5 : TYPICAL MI PATTERN

Tracing 6 : TYPICAL MI PATTERN

Tracing 7 : BRUGADA SYNDROME
Pattern in V_1 & V_2

Tracing 4 : HYPER KALEMIA
Tall peacked T wave in V_3

Tracing 3 : PERICARDITIS

Tracing 2 : LBBB PATTERN

Tracing 1 : LVH (Left Ventricular Hypertrop)

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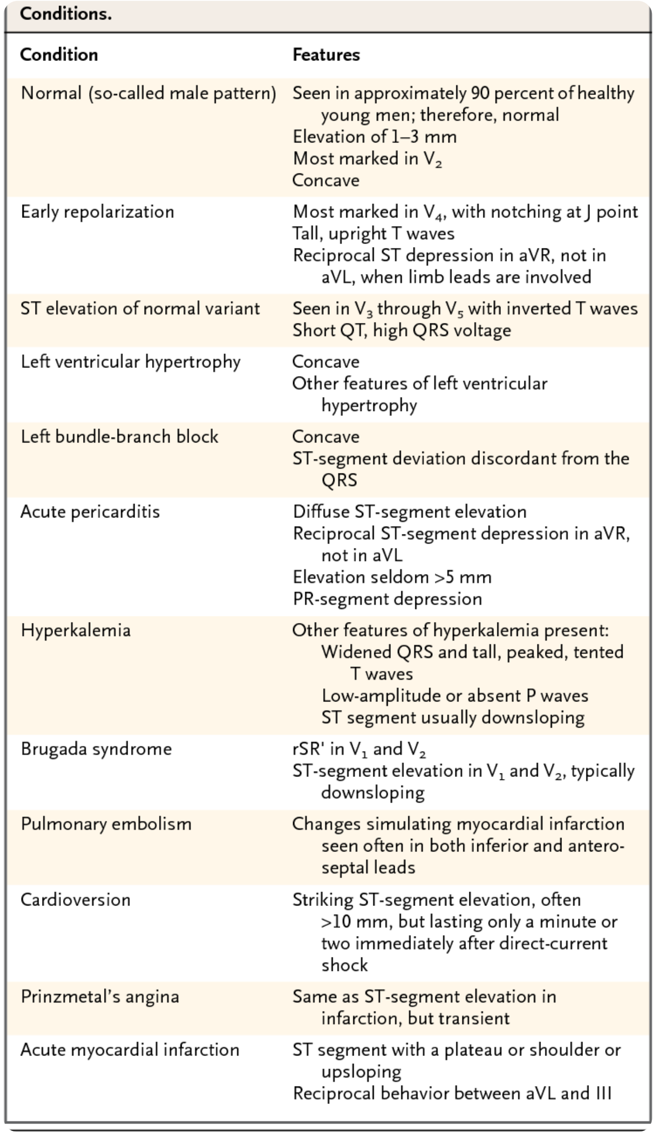
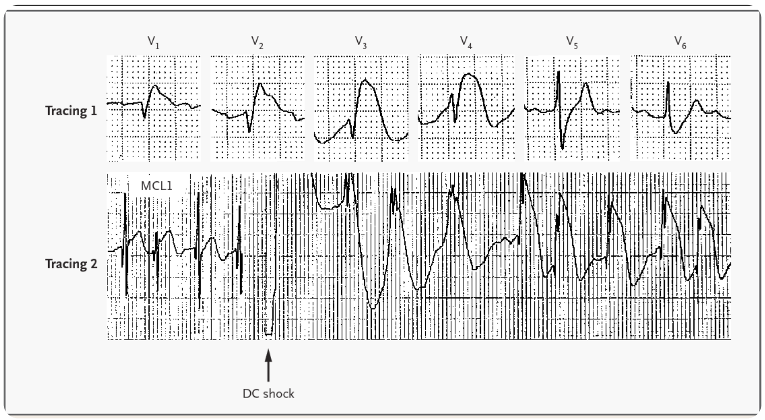


Conditions.

Condition	Features
Normal (so-called male pattern)	Seen in approximately 90 percent of healthy young men; therefore, normal Elevation of 1–3 mm Most marked in V_2 Concave
Early repolarization	Most marked in V_4, with notching at J point Tall, upright T waves Reciprocal ST depression in aVR, not in aVL, when limb leads are involved
ST elevation of normal variant	Seen in V_3 through V_5 with inverted T waves Short QT, high QRS voltage
Left ventricular hypertrophy	Concave Other features of left ventricular hypertrophy
Left bundle-branch block	Concave ST-segment deviation discordant from the QRS
Acute pericarditis	Diffuse ST-segment elevation Reciprocal ST-segment depression in aVR, not in aVL Elevation seldom >5 mm PR-segment depression
Hyperkalemia	Other features of hyperkalemia present: Widened QRS and tall, peaked, tented T waves Low-amplitude or absent P waves ST segment usually downsloping
Brugada syndrome	rSR' in V_1 and V_2 ST-segment elevation in V_1 and V_2, typically downsloping
Pulmonary embolism	Changes simulating myocardial infarction seen often in both inferior and antero-septal leads
Cardioversion	Striking ST-segment elevation, often >10 mm, but lasting only a minute or two immediately after direct-current shock
Prinzmetal's angina	Same as ST-segment elevation in infarction, but transient
Acute myocardial infarction	ST segment with a plateau or shoulder or upsloping Reciprocal behavior between aVL and III



Tracing I : PULMONARY EMBOLISM in V_3 & V_4

Tracing II : PATTERN AFTER DC SHOCK

CAUSES OF ST ELEVATION

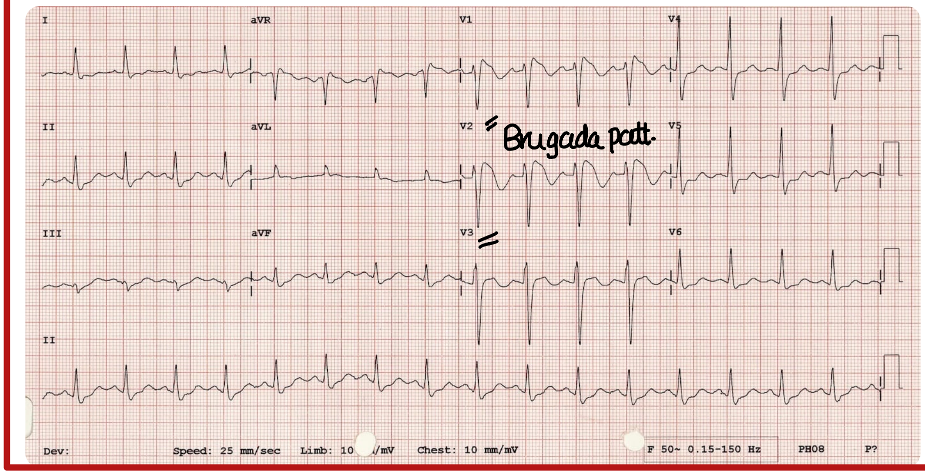
(i) ACUTE M

(ii) PRINMETALS ANGINA

(iii) VENTRICULAR ANEURYSM

(iv) ACUTE PERICARDITIS

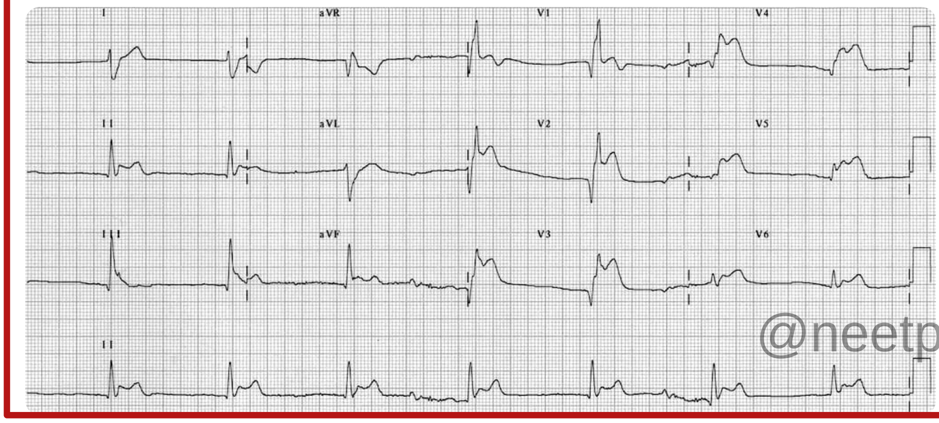
(v) ERS PATTERN



- NO RECIPROCAL CHANGES
- CHANGES ONLY IN V_1 & V_2

↓

BRUGADA PATTERN (prone to sudden cardia death)



- NO RECIPROCAL CHANGES
- Typical convex upward elevation in all leads
- Pt PRESENT WITH CHEST PAIN

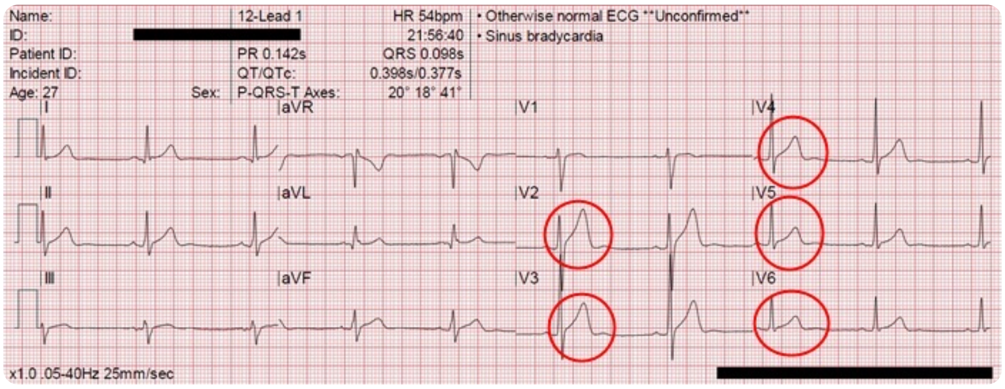
↓

On Angio : NORMAL

called as TAKOTSUBO CARDIO MYOPATHY / STRESS CARDIOMYOPATHY / LVAPICAL BALLOONING SYNDROME

TAKOTSUBOCARDIO MYOPATHY : There is HYOKINESIA of APEX
+
HYPER CONTRACTILE BASE
↓
APICAL BALLOONING OF LV

↓
- * Benign condition
- * Recovers
- * normal angiogram



Name:
ID: [redacted]
Patient ID:
Incident ID:
Age: 27 Sex:
12-Lead 1
PR 0.142s
QT/QTc:
P-QRS-T Axes:
HR 54bpm
21:56:40
QRS 0.098s
0.398s/0.377s
20° 18° 41°
• Otherwise normal ECG **Unconfirmed**
• Sinus bradycardia

x1.0 .05-40Hz 25mm/sec [redacted]

EARLY REPOLARISATION SYNDROME PATTERN

* Slight elevation in J point (or) knotch in J point.

UNIQUE COMPLICATIONS IN MI

1. LV FREE WALL RUPTURE

- seen in ANTERIOR WALL MI (MC)
- can occur at 1-7 days (Generally 3-5 days)
- CARDINAL SIGN : CHEST PAIN
  CARDIAC TEMPONADE
  JVP ELEVATION
  PULSUS PARADOXUS
  ELECTRO MECHANICAL DISSOCIATION
  HYPOTENSION
  CARIOGENIC SHOCK
- CARDIAC TEMPONADE : HYPOTENTION
  MUFFLED HEART SOUNDS
  JVP ELEVATION
  } BECK'S TRIAD

2. PAPILLARY MUSCLE RUPTURE

- Occurs immediately
- Seen in INFERIOR WALL MI (MC)
- Presentation : Pulmonary Edema
- Charachterised by (i) ACUTE ONSET MR
  ↓
  NEW SYSTOLIC MURMUR at APEX
  (ii) V WAVES ON JVP
- TREATMENT : IABP + Surgical Correction
  ↓
  Intra aortic balloon pump

3. VENTRICULAR SEPTAL RUPTURE

- Charachterised by onset within 3-7 days
- Presentation : CHEST PAIN
  SHORTNESS OF BREATH
  HYPOTENSION
- Identified by : (i) ACUTE ONSET MR (New Systolic murmur at Parasternal area)
  (ii) LOUD S2

ONE LINERS

- Killip Classification is used to classify HF in patient with MI
- JVP Elevation is seen in
  RVMI : along with MI
  LV free wall Rupture : 3-5 days after MI
- S_3 GALLOP RHYTHUM : ⟶ SYSTOLIC LV DYSFUNCTION
- S_4 ⟶ DIASTOLIC DYSFUNCTION
- MR is complication of (i) Papillary Muscle rupture
  (ii) Ventricular septal rupture (3 days after MI)
- MRI : MOST SENSITIVE TEST TO DETECT INFARCTION
- CALCIUM CHANNEL BLOCKERS NO ROLE IN ACUTE MI

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HYPERTROPHY & BUNDLE BRANCH BLOCK

SOKOLOW LYON INDEX

- CRITERIA FOR LEFT VENTRICULAR HYPERTROPHY (LVH)

V_1 or V_{2S} + V_5 or V_{6R} > 35 mm

(OR)

R WAVE IN aVL > 11 mm

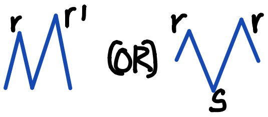
NOTE :

✓ LVH + Left Axis Deviation (LAD)
  (i) In an old patient ⟶ Chronically Hypertensive patient
  (ii) In a 30 yr old ⟶ Hypertensive End-organ damage out of proportion to degree of BP

✓ LVH
- PRESSURE OVERLOAD :
  - CORRESPONDING ST-T CHANGES
  - INVERTED T WAVES & SLIGHT ST DEPRESSION
  - SECONDARY STRAIN PATTERN
- VOLUME OVERLOAD :
  - UPRIGHT T-WAVES

✓ If LVH + Strain + LAD LOOK FOR LBBB

- LBBB PATTERN : r-r' or r-s-r Pattern in V_5-V_6

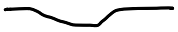


- LBBB MORPHOLOGY : SLIGHTLY WIDE QRS COMPLEX in I, aVL, V_5-V_6

LVH WITH STRAIN V/S MI ST-T CHANGE

LVH WITH STRAIN ↓

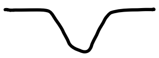
* Assymetrical T wave invertion



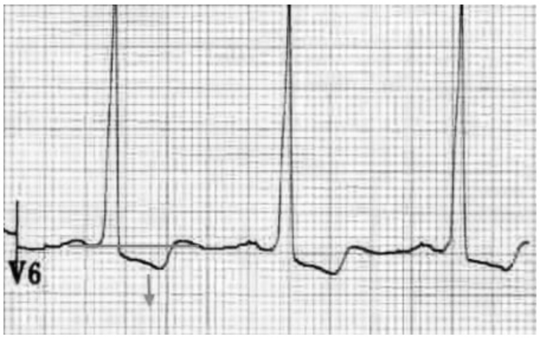
* ST which doesnot dip so much from baseline

MI ST-T CHANGE ↓

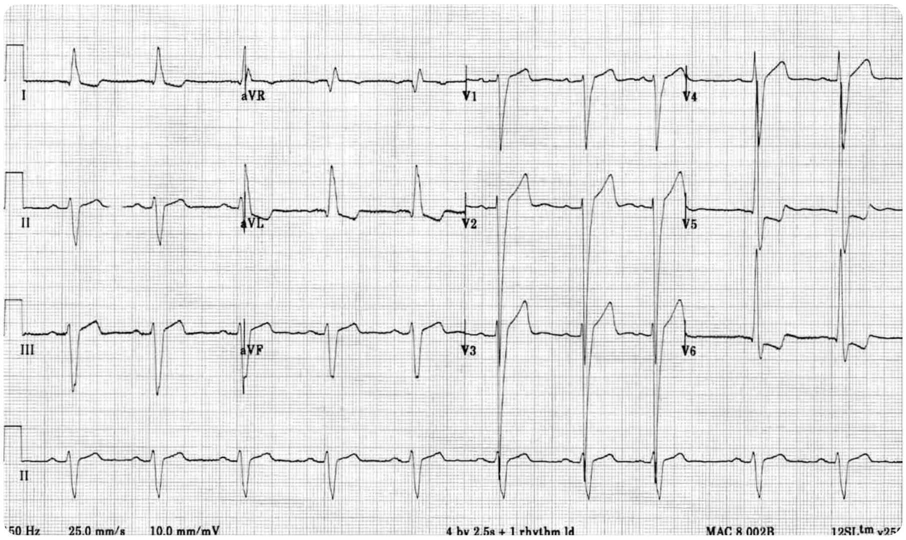
* Symmetrical T wave invertion



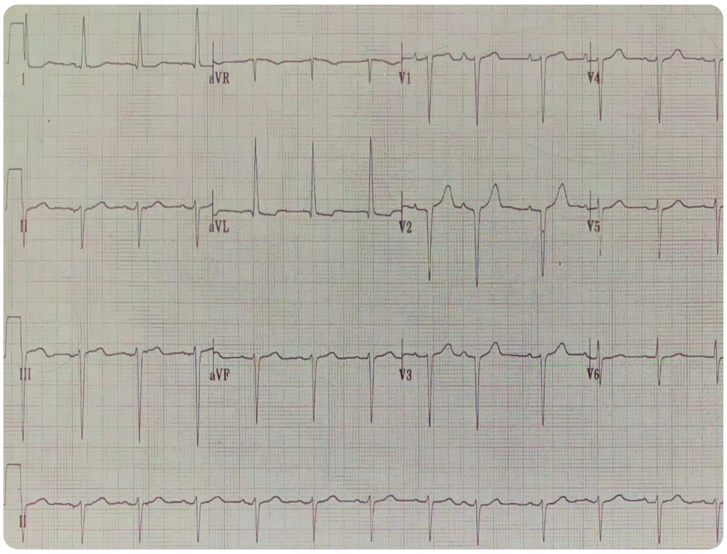
* Severe dip from baseline



LV strain pattern: ST depression and T wave inversion in the lateral leads



- SLIGHTLY WIDE QRS COMPLEX
- AXIS OF EGC : B/W I & aVL ∴ LEFTWARD AXIS
- $V_{1S} + V_{5R}$ > 35mm ∴ LVH
- INVERTED T WAVES & SLIGHT ST DEPRESSION ∴ PRESSURE OVERLOAD
- Slightly WID QRS COMPLEX in I, aVL, V_5-V_6 ∴ LBBB MORPHOLOGY



- NARROW QRS COMPLEX
- LETFT AXIS DEVIATION
- $V_{1S} + V_{5R}$ < 35mm

  But a aVL > 11 mm ∴ LVH
- But there is NO LBBB Morphology

RIGHT VENTRICULAR HYPERTROPHY

- CRITERIA : TALL R WAVE in V_1 i.e >7mm

  (OR)

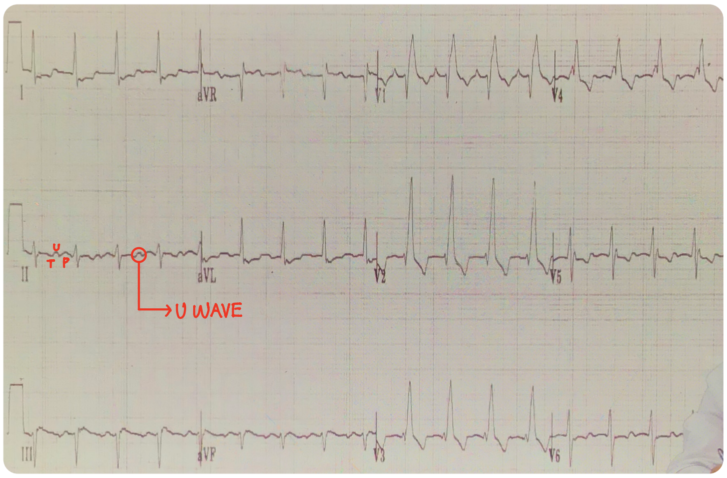
  R/S RATIO = >1
- RBBB MORPHOLOGY : r-r' or r-s-r' Pattern in V_1 - V_2 & w pattern in V_5-V_6

NOTE : Usually LVH is associated with LAD &
RVH is associated with RAD

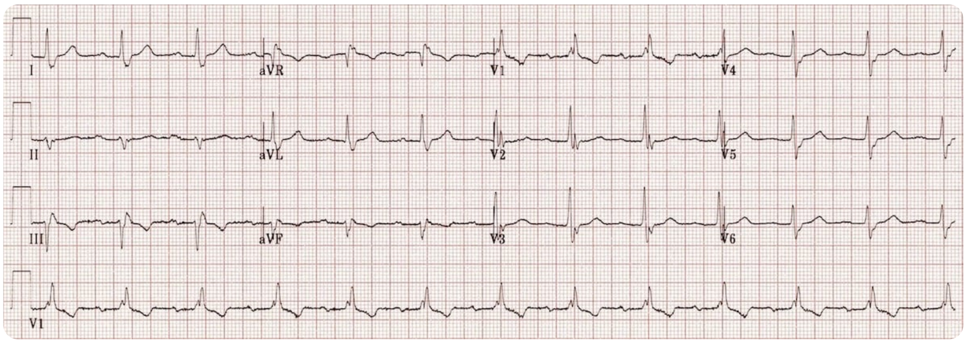
But if RVH + LAD ⟶ BIFASCICULAR BLOCK

→ RBBB + Left Anterior Fascicle Block (or) Left Posterior Fascile Block

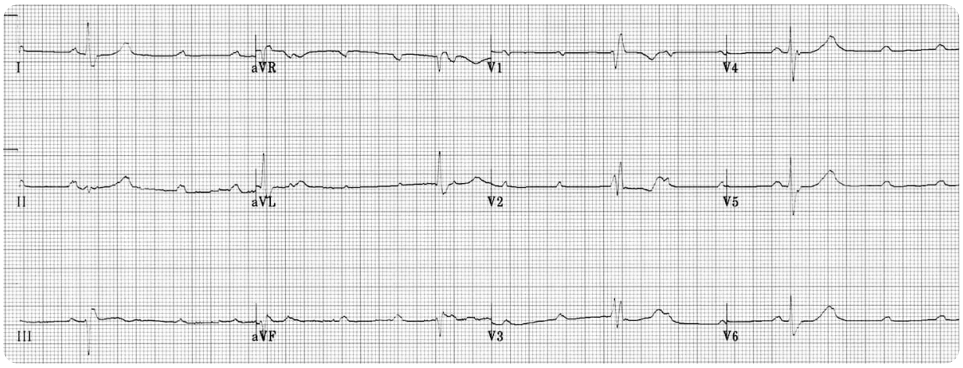
IF PROLONGATION OF PR INTERVAL is called TRIFASCICULAR BLOCK
i.e BIFASCICULAR + 1° AV BLOCK
(or) BIFASCICULAR + 2° AV BLOCK



- NARROW QRS COMPLEX
- PR interval : 4 boxes
- U WAVE is considered significant
  - (i) Height of U > 2/3rd Height of T wave
  - (ii) U & T are discordance
  - → HYPOKALEMIA
- AXIS : LAD
- TALL R WAVE IN V_1 > 7mm
  ∴ RVH.
- RBBB Morphology : M Pattern in V_1 & V_2
- AS RVH + LAD = BIFASCICULAR BLOCK



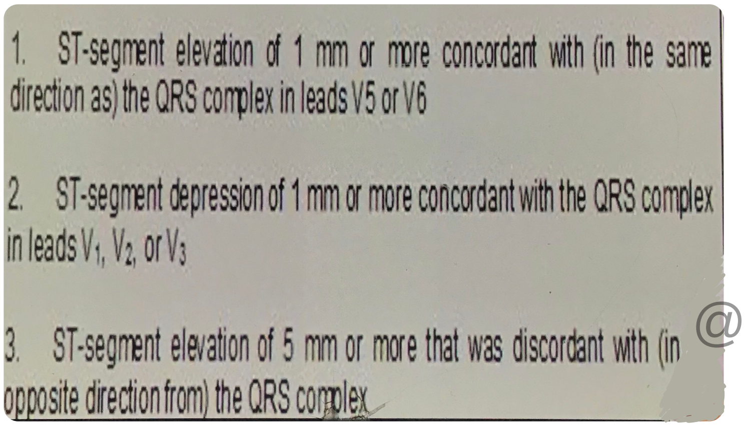
- NARROW QRS COMPLEX
- AXIS : LAD
- Typical RBBB Morphology in V_1–V_2 : BIFASCICULAR BLOCK
- PR PROLONGED
  : TRIFASCICULAR BLOCK



- NARROW QRS COMPLEX
- AXIS : LAD
- Typical RBBB Morphology in V_1–V_2 : BIFASCICULAR BLOCK
- PR PROLONGED
  : TRIFASCICULAR BLOCK

SGARBOSSA'S CRITERIA

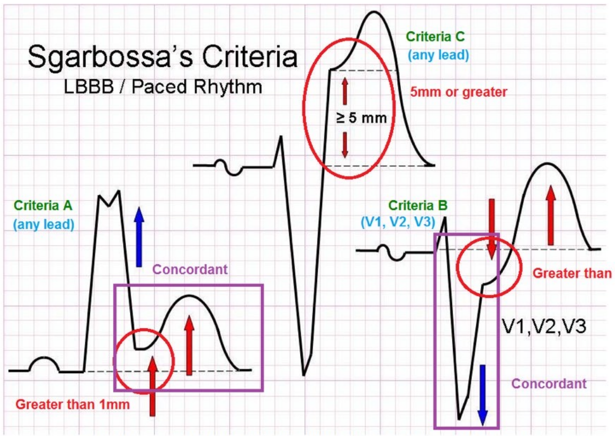
- Help in Diagnosis of MI in presence of LBBB



1. ST-segment elevation of 1 mm or more concordant with (in the same direction as) the QRS complex in leads V5 or V6

2. ST-segment depression of 1 mm or more concordant with the QRS complex in leads V_1, V_2, or V_3

3. ST-segment elevation of 5 mm or more that was discordant with (in opposite direction from) the QRS complex



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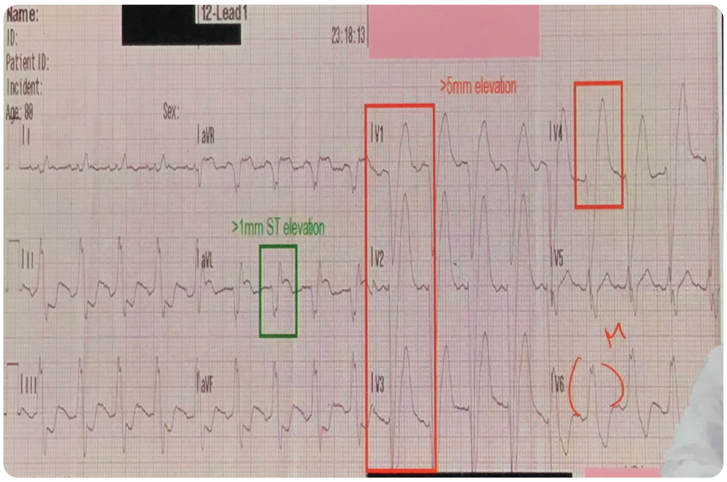


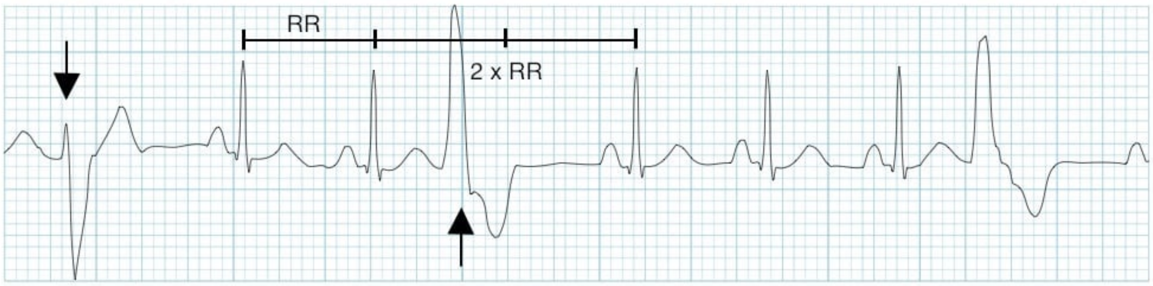
- V_5-V_6 COMPLETE DISCORDANCE
- V_1-V_2 DISCORDANCE
- ST ELEVATION >5mm

↓

LBBB with MI

VENTRICULAR RHYTHUM

VENTRICULAR PREMATURE COMPLEX (VPC)/VENTRICULAR EXTRASYSTOLE



- Pt who is in Normal Sinus Rhythum. Then getting VPC. Then again Normal

- WIDE QRS COMPLEX
- Always followed by COMPENSATORY PAUSE
  i.e the next normal beat arrives after an interval that is equal to double the Preceeding R-R interval
- DISCORDANT ST-T Changes
- COUPLING INTERVAL: Time taken by PVC to occur from the Preceeding QRS Complex

↓

- VPC have CONSTANT COUPLING INTERVAL
- If COUPLING INTERVAL is not constant for a VPC

→ Here VENTRICLE MAY TAKE OVER The RHYTHUM called as VETRICULAR PARASYSTOLE

↓

- It may form fusion complex
- Try to take over rhythum

- INTERPOLATED VPC: VPC BETWEEN TWO SINUS, SUCH IT'S NOT AT ALL ALTERING RHYTHUM

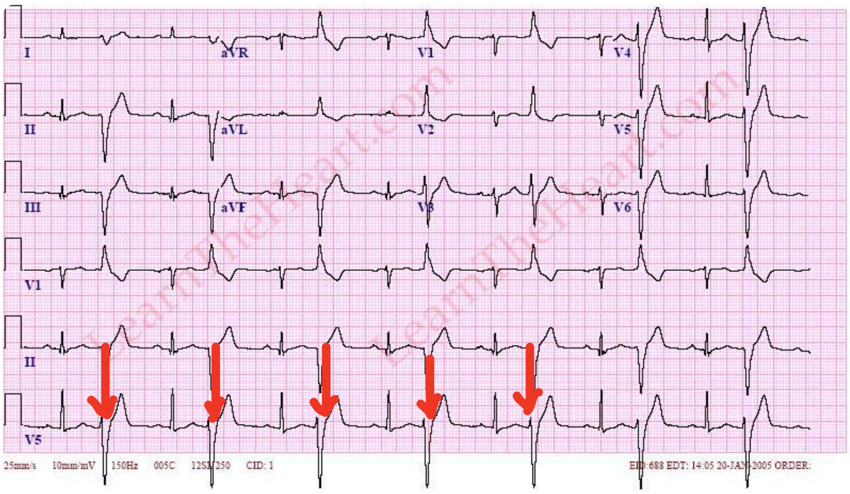
* R ON T PHENOMENON: VPC FALL ON THE T WAVE OF PRECEEDING SINUS IMPULSE

VENTRICULAR BIGEMINY

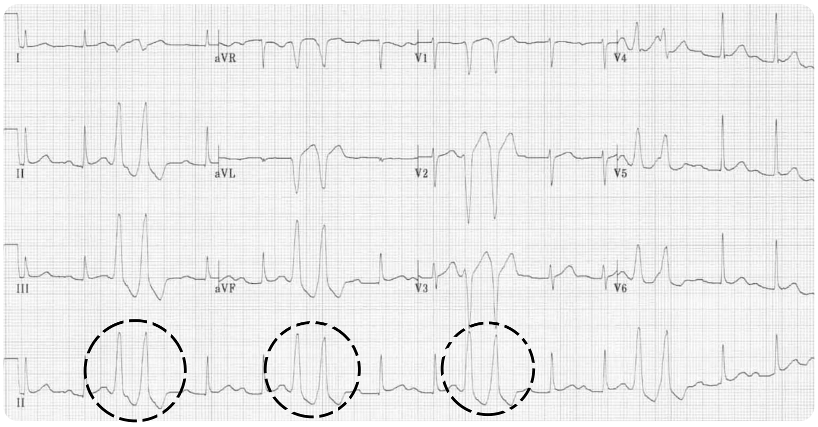
WHEN A VPC ARISES AFTER EVERY SINUS BEAT is called as VENTRICULAR BIGEMINY →

WHEN A VPC ARISES AFTER TWO SINUS BEATS called VENTRICULAR TRIGEMINY

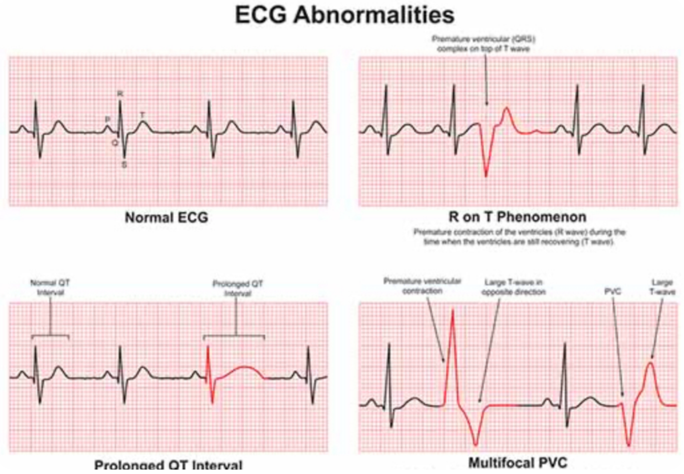
WHEN TWO VPC'S OCCUR TOGETHER CALLED COUPLET


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 150Hz 005C 12SL 250 CID: 1
EID:688 EDT: 14:05 20-JAN-2005 ORDER:

WHEN 3 OR MORE IN A ROW IT'S CALLED VENTRICULAR TACHYCARDIA



Couplet


ECG Abnormalities

Normal ECG

R on T Phenomenon

Premature contraction of the ventricles (R wave) during the time when the ventricles are still recovering (T wave).

Prolonged QT Interval

Multifocal PVC
R
P
T
Q
S
Premature ventricular (QRS) complex on top of T wave
Normal QT Interval
Prolonged QT Interval
Premature ventricular contraction
Large T-wave in opposite direction
PVC
Large T-wave

CLINICAL FEATURES - VPC :
- ASYMPTOMATK
- PALPITATION, MISSED / EXTRA BEAT

TREATMENT :
* ANTI-ARRHYTHMIC IN ISOLATED VT - NO ROLE
* NO ROLE FOR PROPHYLAXIS OF VPC
* GIVE ANTI-ARRHYTHMIC ONLY WHEN VT IS THERE
* BETA BLOCKER IN VPCs IN PATIENT WITH STEMI AND IN PREVENTION OF VF

WHEN VPC'S BECOME DANGEROUS

* USUALLY BENIGN
* FREEQUENTLY OCCURING
* MULTIFOCAL : VPC WITH DIFFERENT MORPHOLOGY
* BIGEMINY
* FIRST TIME OCCURING >40 YRS
* VPC WHICH INCREASES WITH EXERCISE

Usually decreases with Exercise

MINERAL DEFICIENCIES

COPPER DEFICIENCY

A : Anemia (MICROCYSTIC HYPOCHROMIC)
H : HAIR DEFECT
G : GROWTH RETARDATION
E : DEFECTIVE ELASTIN
O : OSTEOPOROSIS
M : MENTAL RETRADATION

CHROMIUM DEFICIENCY :

GLUCOSE INTOLERANCE

PHOSPHORUS DEFICIENCY :

RHABDOMYOLYSIS
HEMOLYSIS
ATAXIA
SEIZURES

ZINC DEFICIENCY

GROWTH RETRADATION
ALOPECIA
AGEUSIA
ANOSMIA
GONDAL DYSFUNCTION
DERMATITIS
IMMUNE DYSFUNCTION

MANGANESE DEFICIENCY

LIPID + CARBOHYDRATE METABOLISM ALTERED
GROWTH & SKELETAL DEVELOPMENTAL RETRADATION
UPPER BODY RASH

IRON DEFICIENCY

IMPAIRED COGNITION
POOR WORK PERFORMANCE
PREMATURE LABOUR
INCREASED PERINATAL DEATH

SELENIUM DEFICIENCY

SEEN IN KESHAN DISEASE
HYPOTHYROIDISM IN SUBHIMALAYAN ZONE

MOLYBDENUM DEFICIENCY

NEUROLOGICAL SYMPTOMS + OESOPHAGEAL CANCER

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TOXICITY/OVERDOSE

COPPER TOXICITY : HEPATOTOXICITY
HEMOLYTIC ANEMIA

BORON OVERDOSE : DEVELOPMENTAL DEFECT
MALE STERILITY
TESTICULAR ATROPHY

IRON TOXICITY : INCREASED SUSCEPTIBILITY TO MALARIA

MANGANESE OVERDOSE : PARKINSON LIKE FEATURES

LEAD TOXICITY : GOUT LIKE FEATURES
∴ CALLED AS SATURNINE GOUT /LEAD NEPHROPATHY
HYPERURICEMIA, TUBULOINTERSTIAL DISEASE

CADMIUM TOXICITY : NEPHROTOXICITY with SEVERE BONE PAIN
COUCH OUCH NEPHROPATHY

CHROMIUM TOXICITY : RENAL FAILURE + LUNG CANCER
↓
TUBULOINTERSTIAL DIS.

ARSENIC TOXICITY : PERIPHERAL NEUROPATHY
GARLIC LIKE ODOUR
LUNG & NASAL CANCERS

FLUORINE TOXICITY : CALCIFICATION OF TENDON/LIGMENT
EXOSTOSES

ZINC TOXICITY : PREDISPOSES TO PULMONARY FIBROSIS

ACID BASE REGULATION

✓ 70,00,000 nmol of H^+ produced by body perday
- Highly Cellulotoxic
- PLASMA CONC. H^+ = 40 nEq/ml (This reduction is done by buffering)

CHEMICAL BUFFER SYSTEM

1. BICARBONATE BUFFER SYSTEM (BBS) MOST EFFICIENT
2. PHOSPHATE BUFFER SYSTEM

} EXTRACELLULAR BUFFER

- pKa → Eg: H_2SO_4, pKa = 2.5
  - ↓ Dissociation constant
  - At this pH it will be
    - 50% → Dissociated (H^+)
    - 50% → Undissociated (AH)
  - But Blood pH = 7.4
  - At this pH, $H_2SO_4 \longrightarrow H^+$ form ∴ can't be a buffer
  - ∴ Buffer pKa should be near to 7.4

- pKa of HCO_3^- = 6.1 (weak acid) : ∵ it's concentration in Blood is high IT CAN ACT AS BUFFER

$$\begin{tikzcd} HCO_3^- + H^+ \arrow[r] & H_2CO_3 \arrow[dl] \arrow[dr] & \\ H_2O & & CO_2 \end{tikzcd}$$

- pKa of HPO_4^{2-} = 6.8 (weak acid)

$$HPO_4^{2-} + H^+ \longrightarrow H_2PO_4^-$$

[BICARBONATE IS MORE EFFICIENT BECAUSE HCO_3^- CONC IS VERY HIGH THAN HPO_4^{2-}]

- INTRACELLUR BUFFER
  1. PROTEINS : MOST IMPORTAN Hb
  2. PHOSPHATE

RESPIRATORY BUFFER SYSTEM

- Exhailing or retaining of CO_2 in responce to extracellular pH

$$HCO_3^- + H^+ \longrightarrow H_2CO_3 \longrightarrow \begin{cases} H_2O \\ CO_2 \end{cases}$$

CO_2 : Exhaled out ; Resp. compensation

NOTE:

Whenever there is Metabolic acidosis : Respiratory compensation is CO_2 being EXHALED OUT

↓

Called as RESPIRATORY ALKALOSIS

RENAL BUFFER SYSTEM

- NON VOLATILE ACIDS PRODUCED/DAY : 80 mEq

↓

EXCRETED BY KIDNEY

- 4320 mEq of HCO_3^- FILTERED/DAY AT GLOMERULUS

↓

This all should be RE-ABSORBED

REABSORPTION OF HCO^{3-} : 80% PCT
10% Thick Ascending Limb
10% DCT

For every molecule of HCO_3^- RE-ABSORBED, ONE H^+ IS EXCRETED.
∴ NEED 4320 mEq H^+

- Total H^+ Excreted/day : ⟶ 4320 + 80 = 4400 mEq
For excretion of 4400 mEq H^+ ⟶ > 2500 Liters of URINE/DAY

BUT CONCENTRATION OF H^+ in URINE ⟶ 0.03 nEq/L
∴ Management of H^+ excretion done by urinary buffers.
(i) AMMONIA BUFFER
(ii) PHOSPHATE BUFFER

NOTE:

- pKA = pH at which ASSOCIATED FORM = DISSOCIATED FORM
i.e HA = A

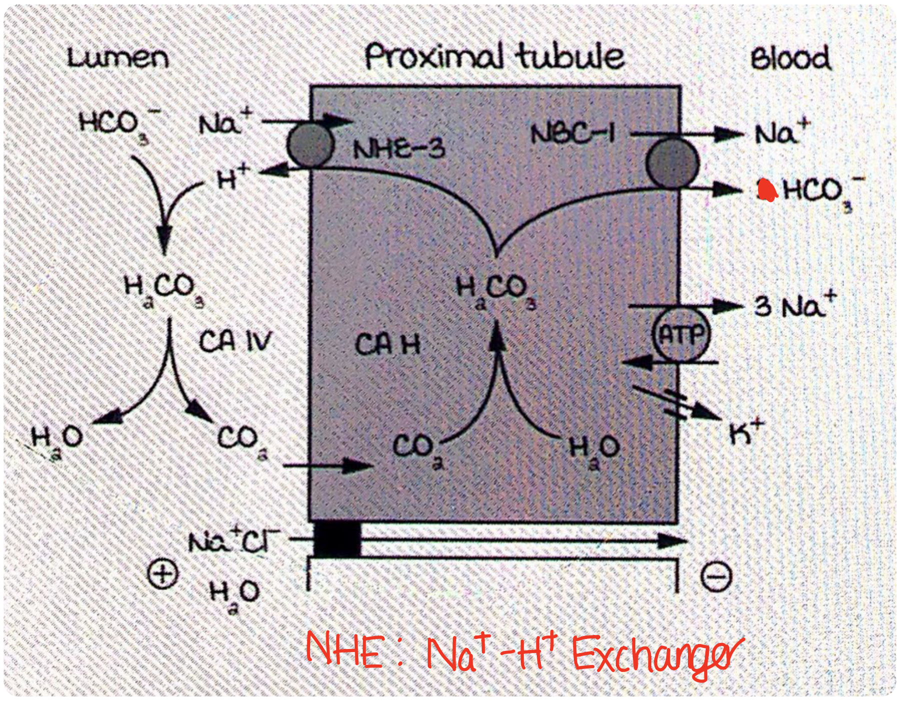
- HANDERSON EQUATION :

$$pH = pKA + \log \frac{A}{HA}$$

- BICARBONATE GENERATION = NET ACID EXCRETION IN URINE = 70-80 meq/day
ACID is getting excreted in the form of : * Ammonium
* Titrable acids (Phosphate/Creatinine/Butyrate)

∴ NAE = NH_4^+ + Titrable acid = 70-80 mEq/day

BICARBONATE AT PROXIMAL TUBULE

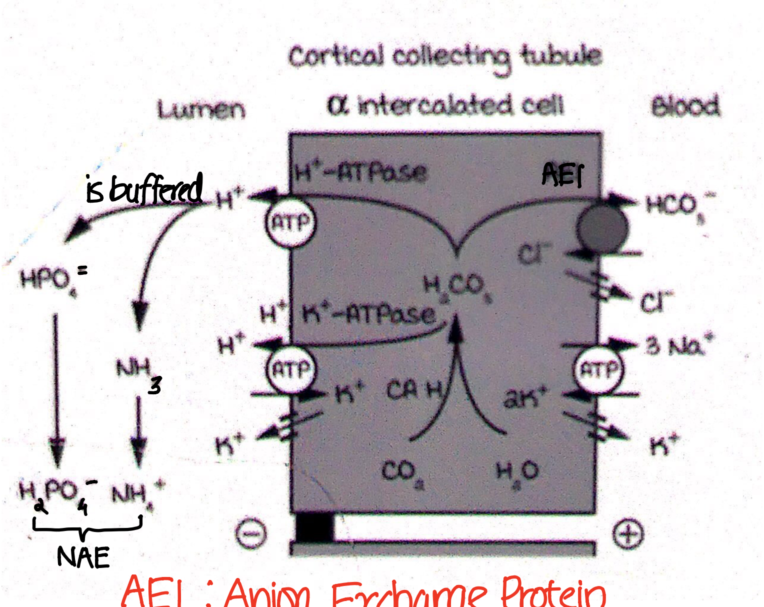


NHE : Na^+-H^+ Exchanger

NOTE:

- EVERY MOLECULE OF HCO_3^- FILTERED INTO LUMEN OF TUBULE, ONE MOLECULE OF H^+ EXCRETED.
- PROBLEM IN NHE-3
  ↓
  BICARBONATE EXCRETION
  ↓
  ∴ SEVERE ACIDOSIS
  PROXIMAL RENAL TUBULAR ACIDOSIS

ACIDIFICATION OF URINE



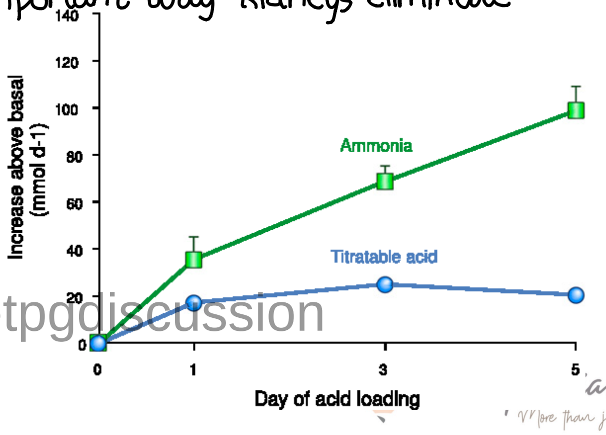
AE1 : Anion Exchange Protein

- ACIDIFYING MACHINERY ⟶ CORTICAL COLLECTING TUBULE (α INTERCALATED CELLS)
- Problem with H^+-K^+ ATPase
  ↓
  DISTAL TUBULAR ACIDOSIS
  (as H^+ can't excrete out)
- H^+ in urine is predominantly from α-intercalated cell

AMMONIA RECYCLING

- Ammonium synthesis & excretion is the most important way kidneys eliminate non volatile acids.
- In Responce to acid loading (From Graph)

  (i) NO TITRATABLE ACID INCREASE
  (ii) AMMONIUM EXCRETION INCREASE
  ↓
  ∴ it's the Ammonium that is most important in acid load.



a.com
More than just notes...

FORMATION OF AMMONIA: INSIDE PCT

GLUTAMINE → GLUTAMATE → α Ketoglutarate

↳ NH_4^+ ↘ ↙ NH_4^+

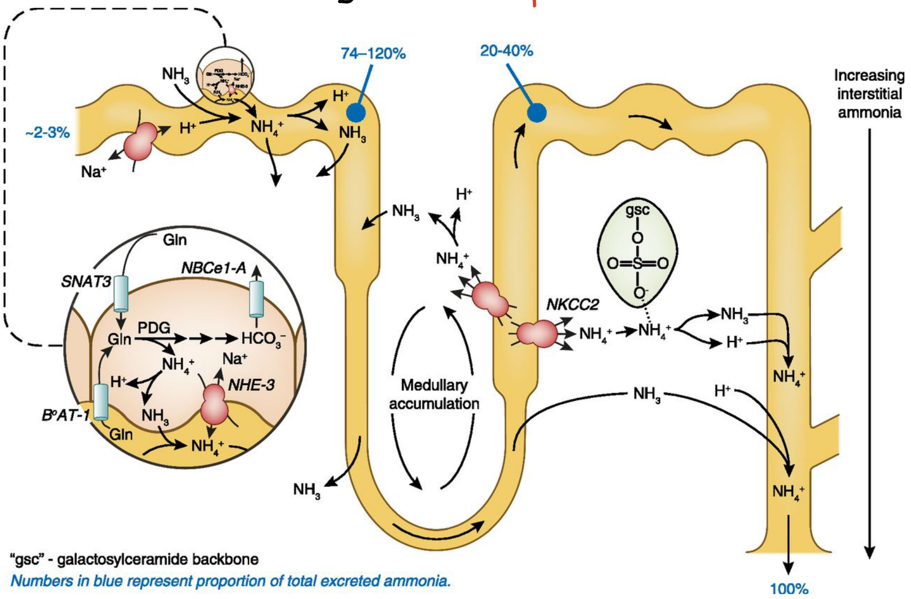
Na^+-H^+ Exchanger in PCT

↓

NH_4^+ EXCRETED INTO LUMEN

↙ NH_3 (Reach Ascending limb) ↘ H^+

- In response to ↑ acid load Kidney compensates by ↑ AMMONIA EXCRETION
- **STIMULI OF AMMONIA PRODUCTION** ⟶
  - METABOLIC ACIDOSIS
  - HYOKALEMIA
- NH_3 at THICK ASCENDING LIMB ⟶ $Na^+K^+2Cl^-$ ⟶ REACH THE INTERSTIUM ↓ COLLECTING DUCT ⟶ URINE ⟶ NH_4^+
- pKa of ammonia ⟶ 9.2 ∴ at PHYSIOLOGICAL pH 98% is NH_4^+
- AMMONIA generated in the Kidney ⟶ NH_4^+



"gsc" - galactosylceramide backbone

Numbers in blue represent proportion of total excreted ammonia.

ACIDS IN THE BODY

- 2 TYPES
  - (i) VOLATILE ACIDS
    - Formed by metabolism of
      - carbohydrates
      - fats
      - proteins
    - Excreted as CO_2 from lungs
  - (ii) NON VOLATILE ACIDS : (fixed acid) : 70-80 mEq/day = 1mEq/kg/day
    - Formed by metabolism
      - Phospholipid
      - Nucleic acids

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- Eg : Sulphuric acids, Phosphoric acid, organic acids
- Excreted by Kidneys

- Henderson Hasselbach's Equation : $pH = pKa + \log \frac{[A^-]}{[HA]}$

- According to Henderson Hasselbach's Equation, important determinants of H^+
  - → Bicarbonate (22-26 mEq/L)
  - → pCO_2 (40mmHg)

First Defence : Chemical Buffer
2nd Defence : Respiratory Buffer
3rd Defence : Renal Buffer

METABOLIC ACIDOSIS

- HEART :
  - ↓ myocardial contractility
  - Sympathetic overactivity
  - ↑ sensitivity to catecholamine
- CNS :
  - Lethargy, Disorientation, Stupor, Coma (HYPOACTIVITY)
- HYPERVENTILATION
- HYPERKALEMIA

- MEASURED CATIONS + UNMEASURED CATIONS = MEASURED ANIONS + UNMEASURED ANIONS

$$MC + UC = MA + UA$$
$$MC - MA = UA - UC$$
$$Na^+ - (Cl^- + HCO_3^-) = UA - UC$$
$$\text{ANION GAP} = UA - UC = 8\text{-}12 \text{ mEq/L}$$

- CORRECTED ANION GAP = ANION GAP + 2.5 (4.5 − S. Albumin)

CLASSIFICATION OF METABOLIC ACIDOSIS :

(i) HIGH ANION GAP MA : (Pneumonic : KUSMAL) → Ethylene Glycol
  - KETOACIDOSIS →
    - DM
    - Alcohol
    - Starvation

    } CAUSES FOR ENDOGENOUS KETOACIDOSIS
  - Lactic acidosis
  - Uremic acidosis
  - Methanol/ethylene glycol
  - Salicylate/Paraldehyde

  } EXOGENOUS ACIDS

(ii) NORMAL ANION GAP MA / HYPERCHLOREMIC METABOLIC ACIDOSIS
  - Renal acidification defect → RTA (TYPE 1 or 2 or 4)
  - GI Loss of HCO_3^- →
    - Diarrhoea
    - Small bowel tumor
    - Ureterosigmoidostomy
    - Anion exchange resins

NOTE : ANION GAP = $Na^+ - (Cl^- + HCO_3^-)$

In ACIDOSIS → ↓ HCO_3^- → Compensated by Cl^- → ∴ N ANION GAP

But if Cl^- not compensates HCO_3^-

↳ Some other molecule compensate

↓

HIGH ANION GAP

METABOLIC ALKALOSIS

- HEART : Cardiac arrhythmias
- BRAIN : Cerebral Insufficiency → HYPOXIA : Lt shift of ODC
- NM SYSTEM : Muscle weakness
- HYPOCALCEMIA
- HYPOKALEMIA
- HYPO MAGNESEMIA

} ∴ LOT OF EXCITABILITY ISSUES

CLASSIFICATION OF M. ALKALOSIS

(i) CHLORIDE/SALINE SENSITIVE/RESPONSIVE :

- ⁎ Urinary Cl^- < 15 mEq/L i.e URINE CHLORINE IS LOW
- ⁎ Vomiting
- ⁎ Nasogastric suctioning
- ⁎ Long term diuretic use
- ⁎ Cystic Fibrosis
- ⁎ Post hypercapnia
- ⁎ Pyloric Stenosis

(ii) CHLORIDE UNRESPONSIVE : SALINE RESISTANT

- ⁎ Urinary Cl^- > 15 mEq/L
- ⁎ with HTN → RAS Activation
  - ↳ Aldosterone tumors
  - Liddle Syndrome
- ⁎ without HTN →
  - • Recent Diuretic Use
  - • Barter syndrome
  - • Gitelman syndrome

CHLORIDE DEPLETION → More Na^+ Delivered distally (Chloride get absorbed along with Na^+)

↓

Contracted ECF volume

↓

⊕ RAS & Aldosterone ←

↓

Na^+ Reabsorption

H^+ - K^+ Excreted

∴ HYPOKALENIA

ALKOLOSIS

ACID BASE REGULATION-2

ABG - ARTERIAL BLOOD GAS CONTRAINDICATION

- Abnormal Modified gas precautions
- Local infection / distorted anatomy at the puncture site
- Severe peripheral vascular disease of artery
- Active Raynaud's Syndrome

ABG - PROCEDURE AND PRECAUTIONS

- Preffered artery : • Radial artery :MOST PREFFERED
  • Femoral artery
- 21 Guge SYRINGE USED

① Flush the syringe with 0.5ml of 1:1000 Heparin
② Don't leave excess heparin in syringe

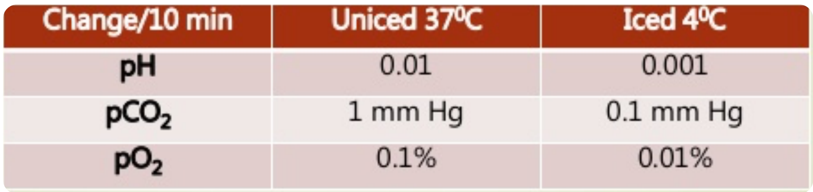
↑ HEPARIN ⟶ ↑ DILUTIONAL EFFECT ⟶ ↓ HCO_3, ↓ PCO_2

③ Ensure no air bubbles

Air bubble = pO_2 mm Hg, pCO_2 0mm Hg

∴ AIR BUBBLE + BLOOD = ↑ pO_2, ↓ pCO_2

④ Early analysis & transported via cold chain

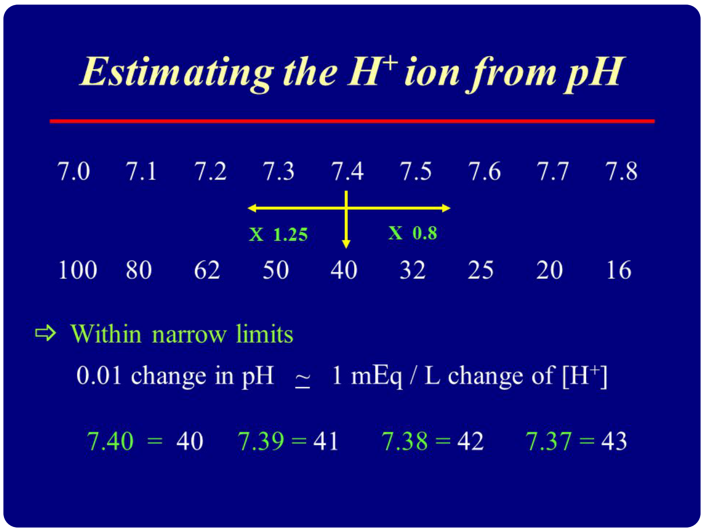


Change/10 min	Uniced 37°C	Iced 4°C
pH	0.01	0.001
pCO_2	1 mm Hg	0.1 mm Hg
pO_2	0.1%	0.01%

ESTIMATION OF H^+ FROM pH

- as pH ↓ ⟶ H^+ ↑

pH 7.4 ⟶ 40
pH 7.3 ⟶ 50
pH 7.2 ⟶ 62
pH 7.5 ⟶ 32



Estimating the H^+ ion from pH

7.0	7.1	7.2	7.3	7.4	7.5	7.6	7.7	7.8
100	80	62	50	40	32	25	20	16

➩ Within narrow limits

0.01 change in pH ~ 1 mEq / L change of $[H^+]$

7.40 = 40 7.39 = 41 7.38 = 42 7.37 = 43

NORMAL VALUES OF ABG

- pH ⟶ 7.36 – 7.44 (7.40)
- HCO_3^- ⟶ 24 – 26 mEq/L (24)
- pCO_2 ⟶ 35 – 45 (40)
- H^+ ⟶ 36 – 44 mEq/L (40nEq/L)
- pH < 7.2 → Severe acidosis
- pH > 7.6 → Severe alkalosis

CALCULATION OF BICARBONATE

STEP I : Obtain ABG & Serum Electrolytes simultaneously
(Validating the ABG)

STEP II : • HCO_3^- / MEASURED HCO_3^- $= 24 \times \frac{pCO_2}{H^+}$

- Measured HCO_3^- is compared with venous blood
- For ABG to be compactible
  The measured HCO_3^- should be within ±2 values compared to HCO_3^- of venous blood

EXAMPLE : ABG :
pH : 7.3 ∴ $[H^+] = 50$
pCO_2 : 25

$$HCO_3^- = \frac{24 \times pCO_2\,(25)}{H^+\,(50)} = 12$$

└→ Serum panel HCO_3^- : 11 ∴ COMPACTIBLE

STEP III : LOOK FOR pH

if pH > 7.4 ⟶ Alkalosis
if pH < 7.4 ⟶ Acidosis
if pH = 7.4 ⟶ Normal or Mixed ABG

STEP IV : Respiratory or metabolic component has changed in the direction of pH

EXAMPLE : I

pH : 7.32 ↓ ⟶ Acidosis
HCO_3^- : 16 ↓ ⟶ Acidosis
pCO_2 : 35 ↓ ⟶ Alkalosis
} METABOLIC ACIDOSIS

II

pH : 7.46 ↑ ⟶ Alkalosis
pCO_2 : 25 ↓ ⟶ Alkalosis
HCO_3^- : 20 ↓ ⟶ Acidosis
} RESPIRATORY ALKALOSIS

III :

pH : 7.42 : ALKALOSIS
HCO_3^- : 28 : ALKALOSIS
pCO_2 : 45 : ACIDOSIS

∴ METABOLIC ALKALOSIS

IV :

pH : 7.46 : Alkalosis
pCO_2 : 25 : Alkalosis
HCO_3^- : 16 : Acidosis

∴ Respiratory alkalosis

STEP IV : COMPENSATION

1. COMPENSATION FOR METABOLIC ACIDOSIS

- By Respiratory alkalosis
- $HCO_3 \downarrow$ by 1mEq/L = $pCO_2 \downarrow$ 1.2mmHg

EXAMPLE I :

pH : 7.10 : ACIDOSIS
pCO_2 : 32 : ALKALOSIS
HCO_3^- : 10 : ACIDOSIS
} METABOLIC ACIDOSIS

Here HCO_3 fall from 24 ⟶ 10 = 14mEq/L fall
∴ COMPENSATION = 14 × 1.2
= 17
∴ The expected pCO_2 = 40 − 17 = 23
∴ It's UNCOMPENSATED or PARTIALLY COMPENSATED METABOLIC ACIDOSIS
∵ problem pCO_2 is 32 & the expected is 23

EXAMPLE II :

pH : 7.1 : ACIDOSIS
pCO_2 : 32 : ALKOLOSIS
HCO_3^- : 12 : ACIDOSIS
MET. ACIDOSIS
24 − 12 = 12 mEq/L
12 × 1.2 = 14.4
∴ Expected pCO_2 = 40 − 14.4
≘ 25
∴ UNCOMPENSATED/
MET. ACIDOSIS WITH MILD RESP. ALKOLOSIS

EXAMPLE - III

pH = 7.35 : ACIDOSIS
HCO_3^- = 14 : ACIDOSIS
pCO_2 = 28 : ALKALOSIS
∴ Metabolic acidos
Fall = 10
∴ expected fall in pCO_2 = 10 × 1.2 = 12
∴ expected pCO_2 = 40 − 12
= 28
∴ COMPENSATED/
SIMPLE METABOLIC ACIDOSIS/
MET. ACI with RESP. ALKOLOSIS

EXAMPLE IV :

pH : 7.1 : Acidosis
pCO_2 : 45 : Acidosis
HCO_3^- : 10 : Acidosis

METABOLIC + COMBINED ACIDOSIS

EXAMPLE V :

pH : 7.1 : Acidosis
pCO_2 : 35 : Alkalosis
HCO_3^- : 10 : Acidosis
METABOLIC ACIDOSIS
Fall in HCO_3^- = 14
∴ expected fall in pCO_2 = 14 × 1.2
= 17
∴ expected pCO_2 = 40 − 17
= 23
∴ UNCOMPENSATED

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ACID BASE REGULATION-3

COMPENSATION : STEP IV

2. COMPENSATION FOR METABOLIC ALKALOSIS

HCO_3^- ↑ by 1 mEq/L = pCO_2 ↑ by 0.7 mmHg

EXAMPLE 1 :

pH = 7.45 : Alkalosis
HCO_3^- = 32 : Alkalosis
pCO_2 = 40 : Normal

∴ Metabolic Alkalosis

Increase in HCO_3^- = 32 − 24 = 8
∴ Expected ↑ in pCO_2 = 8 × 0.7 = 5.6

∴ Expected pCO_2 = 40 + 5.6 = 45.6 mmHg

∴ UNCOMPENSATED METABOLIC ALKALOSIS

NOTE :

pH = 7.30 Acidosis
HCO_3^- = 14 Acidosis

pCO_2 = 20
↓
Met. Acidosis
+
Resp. alkalosis

pCO_2 = 28
↓
Metabolic Acidosis
Fully compensated
or
Simple metabolic acidosis

pCO_2 = 35
↓
Metabolic acidosis
Partially compensated

pCO_2 = 42
↓
Met + Respiratory
Acidosis

WINTERS FORMULA for METABOLIC ACIDOSIS

$$pCO_2 = 1.5 \times HCO_3^- + 8 \pm 2$$

EXAMPLE

pH = 7.3
HCO_3^- = 16
pCO_2 = 30

$pCO_2 = 1.5 \times 16 + 8 \pm 2$
$= 32 \pm 2$
$= 30 - 34$

3. COMPENSATION FOR RESPIRATORY ACIDOSIS & ALKALOSIS

- Respiratory

1 4 → RESP. ACIDOSIS

2 4 → RESP. ALKALOSI

- RESP. ACIDOSIS

  - Acute Resp. acidosis → $pCO_2 \uparrow$ 10 mmHg = $HCO_3 \uparrow$ 1mEq/L
  - Chronic Resp. acidosis → $pCO_2 \uparrow$ 10 mmHg = $HCO_3 \uparrow$ 4mEq/L

EXAMPLE : Acute Resp. Acidosis

$pH = 7.25$ Acidosis
$pCO_2 = 55$ Resp. acidosis
$HCO_3^- = 30$ Met. alkalosis
} Resp. acidosis

$pCO_2 \uparrow = 55 - 40 = 15$

$\therefore$ for 10 ↑ → $HCO_3 \uparrow$ by 1

15 ↑ → 1.5

$\therefore HCO_3^- = 24 + 1.5 = 25.5$

$\therefore$ It's Respiratory acidosis + Metabolic alkalosis

if $HCO_3^- = 24$ → UNCOMPENSATED RESP ACIDOSIS

- RESP AIKALOSIS

  - ACUTE RESP. ALKALOSIS → $pCO_2 \downarrow$ 10mmHg = $HCO_3^- \downarrow$ 2mEq/L
  - CHRONIC RESP. ALKALOSIS → $pCO_2 \downarrow$ 10mmHg = $HCO_3^- \downarrow$ 4mEq/L

EXAMPLE : Acute

$pH = 7.46$ Alkalosis
$pCO_2 = 30$ Resp. Alkalosis
$HCO_3^- = 22$ Met. Acidosis
} Respiratory alkalosis

$pCO_2 \downarrow = 10$ mmHg

$\therefore \downarrow HCO_3^- = 2$

$\therefore HCO_3^- = 24 - 2 = 22$

$\therefore$ COMPENSATED RESP. AILKALOSIS

EXAMPLE :

A 40 yr old man is admitted for diarrhea & breathlessness for 2 days

$pH = 7.28$ $pCO_2 = 29$ $pO_2 = 100$ $HCO_3^- = 12$

$Na^+ = 138$ $K^+ = 2.9$ $Cl^- = 115$ Urea = 70

S.Cr = 1.4 Sugar = 110

Ans :

pH	: 7.28	Acidosis	} Metabolic acidosis
pCO_2	: 29	Alkalosis	
HCO_3^-	: 12	Acidosis	

Fall is by 12, Expected fall $= 12 \times 1.4 = 14.4$

$\therefore$ Expected $pCO_2 = 40 - 14.4 \approxeq 25$

$\therefore$ Partly compensated

CALCULATION OF ANION GAP : STEP V

- Serum anion gap $= Na^+ - (Cl^- + HCO_3^-) = 8\text{-}12$ mEq/L
- Metabolic acidosis ⟶
  - Loss of HCO_3^-
  - If loss of HCO_3^- compensated by Cl^-

    Normal anion gap metabolic acidosis
- When loss of HCO_3^- is compensated by an ion other than Cl^- (Unmeasured anion) ⟶ High anion gap metabolic acidosis

EXAMPLE :

A 40 yr old man is admitted for diarrhea for 1 week & breathlessness for 1 day

$pH = 7.3$ $pCO_2 = 30$ $pO_2 = 100$ $HCO_3 = 16$

$Na^+ = 140$ $K^+ = 6.9$ $Cl^- = 102$ Urea = 120

S.Cr = 5.4 Sugar = 110

Ans : → Here Metabolic acidosis

fall in $HCO_3^- = 8$

$8 \times 1.2 = 9.6$ fall in pCO_2

$\therefore$ compensated Met. acidosis

→ Anion gap $= Na^+ - (Cl^- + HCO_3^-)$

$= 140 - (102 + 16)$

$= 22$ High Anion Gap Met acidosis

→ $K^+ = 6.9$ → Severe Hyperkalemia

→ Creatinine high

$\therefore$ Renal failure

→ $\therefore$ HAGMA + Renal Failure + Hyperkalemia

Diagnosis : Uremic acidosis

- In High anion gap metabolic acidosis calculate delta ratio

$$\text{Delta Ratio} = \frac{\text{Delta AG}}{\text{Delta } HCO_3^-} = \frac{\Delta AG}{\Delta HCO_3^-}$$
$$= \frac{AG - 12}{24 - HCO_3^-}$$

Normal = 1-2 → High anion gap Met. Acidosis

Delta Ratio <1 → High anion gap Met. acidosis + Normal anion gap Met. acidosis

Delta Ratio >2 → High anion gap Met. acidosis + Met. Alkalosis

EXAMPLE :

A 40 yr old man is admitted for diarrhea for 1 week & breathlessness for 1 day

pH = 7.3 pCO_2 = 30 pO_2 = 100 HCO_3 = 16

Na^+ = 140 K^+ = 6.9 Cl^- = 102 Urea = 120

S.Cr = 5.4 Sugar = 110

→ Anion gap = $Na^+ - (Cl^- + HCO_3^-)$
= 140 − (102 + 16)
= 22 = High Anion Gap Met acidosis

Δ AG = 22 − 12
= 10

→ Delta Ratio = $\frac{22-12}{24-16}$
= $10/8$ = $\underline{\underline{1.2}}$

∴ Clear high anion gap Metabolic acidosis

EXAMPLE

? A 45 yr old female is admitted for high fever & breathlessness, diagnosed to have acute Cholecystitis

pH = 7.3 pCO_2 = 12 pO_2 = 100 HCO_3^- = 6

Na^+ = 140 K^+ = 5 Cl^- = 105 Urea = 45

S.Cr = 1.6 Sugar = 120

Ans: → Metabolic acidosis
fall in HCO_3^- = 18
18 × 1.2 = 20 = ↓ pCO_2
pCO_2 = 20
∴ well compensated
∴ Met. Aci + Resp. alkalosis

→ Anion gap : 140 − (105 + 6) = 29 (HAGMA)

→ Δ Ratio = $\frac{29-12}{24-6}$
= $17/18$ = <1

∴ HAGMA + NAGMA + Resp. alkalosis

→ Sepsis & producing Lactic acidosis

ACID BASE REGULATION-4

URINE ANION GAP

- To differentiate normal AG Met·acidocis due to RTA or GI loss, urine AG done

URINE AG = Urine Na^+ + (Urine K^+ − Urine Cl^-)

Generally it's positive

Positive urine AG ⟶ RTA
Negative urine AG ⟶ GI loss of HCO_3^-

EXAMPLE

? A 50 yr old known COPD Pt, with cor pulmonale on t/t with furesemide & admited for for severe vomitting.

$pH = 7.41$ $pCO_2 = 80$ $pO_2 = 40$ $HCO_3^- = 48$
$Na^+ = 140$ $K^+ = 4$ $Cl^- = 105$ Urea = 45
S.Cr = 1.2 Sugar = 128

Ans : pH = 7·41 ⟶ put Alkalosis
∴ METABOLIC ALKALOSIS
Rise in HCO_3^- by = 24
exp. Rise in pCO_2 = 24 × 0·7
≏ 17

∴ Rise is = 40 + 17 = 57
∴ RESPIRATORY ACIDOSIS
→ COPD ⟶ Resp. acidosis
→ Vomitting & diuretic use ⟶ Met· Alkalosis

EXAMPLE

? Based on the following information how would you describe the metabolic disorder

ABG : 7·24/42/99 100% O_2 sat on room air
Lytes : Na^+ 143 mEq/L , K^+ 4.0 mEq/L , Cl^- 100 mEq/L , HCO_3^- 21 mEq/L

Ans :→ pH = 7·24 ACIDOSIS
HCO_3^- = 21 ACIDOSIS
pCO_2 = 42 ACIDOSIS
} METABOLIC + RESPIRATORY ACID.

→ Anion Gap = 143 − (100 + 21)
= 22 ⟶ HAGMA + Respiratory ACID.

→ Δ Ratio $= \frac{22-12}{24-21} = \frac{10}{3} = >3$ ⟶ HAGMA + Resp acid + Met·Alkol.

EXAMPLE

? A 10 year old teenager is found unconcious in his home, with an empty medicine bottle in hand. He is taken to ER, Based on following information what is the primary disturbance & does there appear to be appropriate compensation?

ABG: 7.21/42/90 100% O_2 saturation on air HCO_3^- 14 mEq/L

Ans: pH: 7.21
HCO_3^-: 14
pCO_2: 42
} Metabolic acidosis + Respiratory acidosis

EXAMPLE

? A 10 year old pregnant insulin dependent diabetic patient was admitted with a h/o polyuria & thirst. She now felt ill & presented to hospital. There was h/o poor compliance with medical therapy.

- She was afebrile. Chest was clear. Circulation was adequate.
  URINANALYSIS : 2+ Ketones, 4+ glucose
- Na^+ 136, K^+ 4.8, Cl^- 101, pH = 7.26, pCO_2 16 mmHg, pO_2 128 mmHg, HCO_3^- 7.1 mmol/l

ANS:

pH = 7.26 Acidosis
HCO_3^- = 7.1 Alkalosis
pCO_2 = 16 Acidosis
} Metabolic Acidosis

fall in HCO_3^- = 17
expected pCO_2 = 40 − (17 × 1.2)
= 20
} Resp. alkalosis

AG = 136 − (101 + 7)
= 28
} HAGMA (Due to Diabetic Ketoacidosis)

$$\Delta \text{Ratio} = \frac{28-12}{24-7} = \frac{16}{17} = <1$$
} HAGMA + NAGMA

↓

Due to RTA due to Diabetes
(not clear)

EXAMPLE

? A 60yr old woman was admitted with lobar pneumonia. She was on thiazide diuretic for 0 months following a previous admission with congestive cardiac failure. The ABG:

K^+: 2.1, pH: 7.64, pCO_2: 32 mmHg, pO_2: 75 mmHg, HCO_3^-: 33 mmol/l

pH ↓ Alkalosis; pCO_2 ↓ Alkalosis; HCO_3^- ↓ Alkalosis

∴ Metabolic + Resp. Alkalosis (Chronic thiazide use → Metab. Alkal.)

EXAMPLE

? 21 year old Primigravida with hyperemesis.

pH = 7·55, HCO_3^- = 30 mmol/L, pCO_2 = 25mmHg

↓ Alkalosis ↓ Alkalosis ↓ Alkalosis

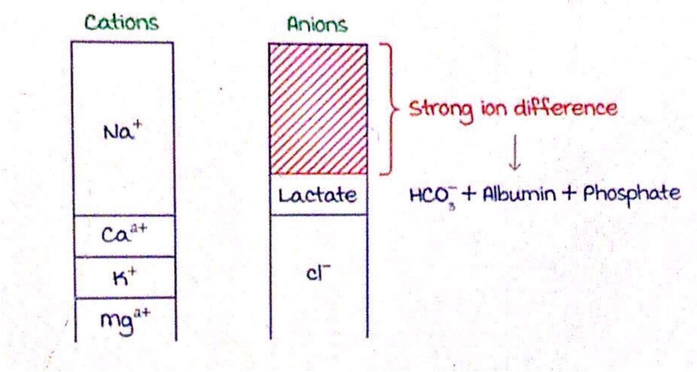
Resp + Metabolic Alkalosis

↓ Hyperventillation ↓ Primigravida

STRONG ION DIFFERENCE

STRONG ION DIFFERENCE = $(Na^+ + K^+ + Mg^{2+}) - (Cl^- + Lactate^-)$

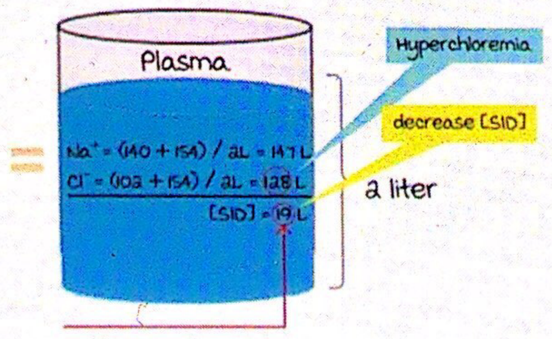
- Difference between measured strong cations & anions
- NORMAL SID ⟶ 40mEq/L



SID = HCO_3^- + Albumin + phosphate

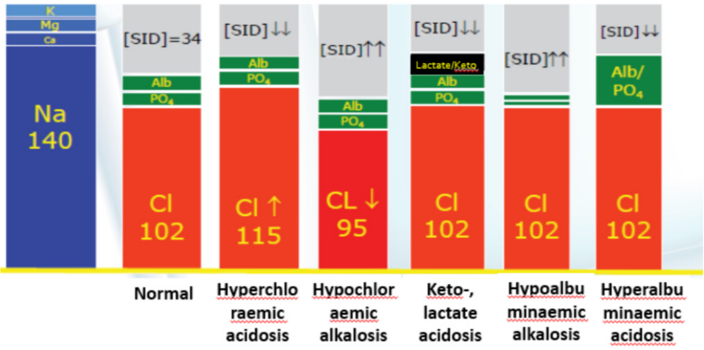
MET. ALKALOSIS ⟶ SID ↑
MET. ACIDOSIS ⟶ SID ↓
DEHYDRATION ⟶ SID ↑
LACTIC ACIDOSIS ⟶ SID ↓
HYPO ALBUMINEMIA ⟶ SID ↓

Saline cause acidosis by decreasing [SID] due to the hyperchloremic


Plasma
Hyperchloremia
decrease [SID]
2 liter
Na^+ = (140 + 154) / 2L = 147 L
Cl^- = (102 + 154) / 2L = 128 L
[SID] = 19 L

[SID] = 19↓ ⟶ pH↓ ⟶ more acidosis

Abnormalities in SID and Weak Acid



NOTE : Adding Ringer Lactate to plasma

Na^+ = (140 + 137)/2 = 139
Cl^- = (102 + 109)/2 = 105
after adding Ringer SID = 34 ⟶ Cause acidosis
initially = 140 - 102 = 38 But less than that of saline

NOTE : Base excess of −2 to +2 is normal
−2, −3, −4 etc ⟶ Met acidosis
> +2 ⟶ Met alkalosis

SID = 40mEq/L
Whatever it decreases from 40 is Base excess negative : MET. ACIDOSIS
Whatever it increases from 40 is Base excess positive : MET. ALKALOSIS

OSMOLARITY

- Calculated osmolarity $= 2Na^+ + \frac{B.Glucose}{18} + \frac{BUN}{2.8} = 285-290$
- Measured osmolarity by Osmometer

OSMOLAL GAP = Measured OSMOLARITY − Calculated Osmolarity = > 10

└⟶ in
- Methanol poisoning
- Ethylene Glycol poisoning

- Diagnosis : High anion gap metabolic acidosis + metabolic alkalosis + Respiratory acidosis

PROBLEM-1

? A young women presented with a h/o watery diarrhea two day after railway journey. She is febrile, tachycardia, has dry mouth & cold extremities Her BP was 90/60 mmHg.

INVESTIGATIONS

Blood Urea	40mg/dl
S. Creatinine	1.4mg/dl
S. Na^+	135mmol/L
S. K^+	2.2 mmol/L
S. Cl^-	108 mmol/L
S. HCO_3^-	18 mmol/L
Anion gap	9 mmol/L
Albumin	2.5g/dL
Corrected AG	$9+2\times(4.5-2.5)=13$

ABG ANALYSIS

pH	7.31
pCO_2	3.5 mmHg
pO_2	95 on room air
HCO_3^-	16 mmol/L
O_2 sat	95%

Ans :
- pH : 7.31 Acidosis
- HCO_3^- : 18 Acidosis
- pCO_2 : 3.5 Alkalosis

} 1° disorder Metabolic acidosis

- pCO_2 expected $= 40-(6\times1.2) = 33$ ⟶ Respiratory alkalosis
- AG = 9 : Normal ; Corrected AG = 13 : HAGMA
- Δ Ratio $= \frac{14-12}{24-18} = 2/6 = \underline{\underline{<1}}$ ⟶ HAGMA + NAGMA + Resp alkalosis

↓ BP 90/60, Shock, Lactic acidosis
↓ Diarrhoea
↓ Hyperventilation

PROBLEM-II

? An insulin dependent diabetic patient stopped taking insulin for 2days & presented to causality with vomiting & breathlessness. He complains of severe thirst. On examination his BP was 100/70 mm Hg, pulse rate 110/min regular & had acidotic breathing. Systolic examination was otherwise unremarkable. Clinical diagnosis : Patient has diabetic ketoacidosis

INVESTIGATIONS

Blood Urea	: 300 mg/dl
S. Creatinine	: 1.4 mg/dl
S. Na^+	: 135 mmol/L
S. K^+	: 5 mmol/L
S. Cl^-	: 100 mmol/L
S. HCO_3^-	: 12 mmol/L
Anion gap	: 23 mmol/L
Albumin	: 3.5 g/dL
Corrected AG	: 25
Urine Ketones	: positive

ABG ANALYSIS

pH	: 7.25
pCO_2	: 27 mmHg
pO_2	: 81 on room air
HCO_3^-	: 12 mmol/L
O_2 sat	: 95%

Ans : HAGMA + Compensated Metabolic acidosis

Δ Ratio $= \frac{25-12}{24-12} = \frac{13}{12} = 1.2 \rightarrow$ HAGMA

No indication to give HCO_3^-

NOTE : if < 7.1 plan for correction of HCO_3^-

Bicarbonate deficit $= [24 - HCO_3^-] \times 0.6 \times$ Body weight

1 amp of HCO_3^- contain = 10 ml

1 ml = 1.7 mEq

10 ml = 17 mEq

50% of deficit should correct in 24 hrs

PROBLEM-III

? A 65 year old woman who has been diabetic & HTN for 5 years on oral hypoglycemic agents & indapamide developed cellulitis of the foot following injury 15 days prior to admission. She was admitted with complaints of muscle weakness. On examination patient was hypovolemic BP 110/70 mm Hg, Hypotonia, with grade 4 muscle power in all four limbs.

INVESTIGATIONS

Blood Urea	: 360 mg/dl
S. Creatinine	: 1.2 mg/dl
S. Na^+	: 132 mmol/L
S. K^+	: 2.6 mmol/L
S. Cl^-	: 82 mmol/L
S. HCO_3^-	: 18 mmol/L
Anion gap	: 32 mmol/L
Albumin	: 4.5 g/dL

ABG ANALYSIS

pH	: 7.41
pCO_2	: 32 mmHg
pO_2	: 71 on room air
HCO_3^-	: 20 mmol/L
O_2 sat	: 95%

Corrected AG : 32

Ans : Respiratory alkalosis (Indapemide -diuretic)
expected $HCO_3 = 22$: Met-acidosis
↓
AG : HAGMA (Hypovolemia & Sepsis)
↓
Δ Ratio = $\frac{32-12}{24-18} = \frac{20}{6} = >2$: Met. alkalosis also

PROBLEM IV

? A 75 yr old man with a longstanding h/o heavy smoking is bought to hospital in a febrile & confused state. He rapidly becomes drowsy. spO_2 is 80%. Clinical assessment indicates the patient is likely to have chronic resp. acidosis. He is hypoxic, so there is possibility of lactic acidosis in addition

INVESTIGATIONS

Blood Urea : 360 mg/dl
S. Creatinine : 1.2 mg/dl
S. Na^+ : 132 mmol/L
S. K^+ : 2.6 mmol/L
S. Cl^- : 82 mmol/L
S. HCO_3^- : 18 mmol/L

ABG ANALYSIS

pH : 7.21
pCO_2 : 70 mmHg
pO_2 : 55 on room air
HCO_3^- : 26 mmol/L
O_2 sat : 80%

Ans : Respiratory + Metabolic acidosis
↓ due to smoking
AG : HAGMA (Sepsis & Lactic acidosis)
Δ Ratio = $\frac{20}{6} = >2$: HAGMA + Met. alkalosis
↓ Cause not clear

PROBLEM V

? A 50 yr old man was admitted with a 3 day h/o abdominal pain & vomitting. The patient was pale & tachypneic, BP was not recordable, & abdomen was rigid. He was resuscitated with IV fluids & take up for emergency laprotomy which revealed infected ascitic fluid & perforated duodenal ulcers

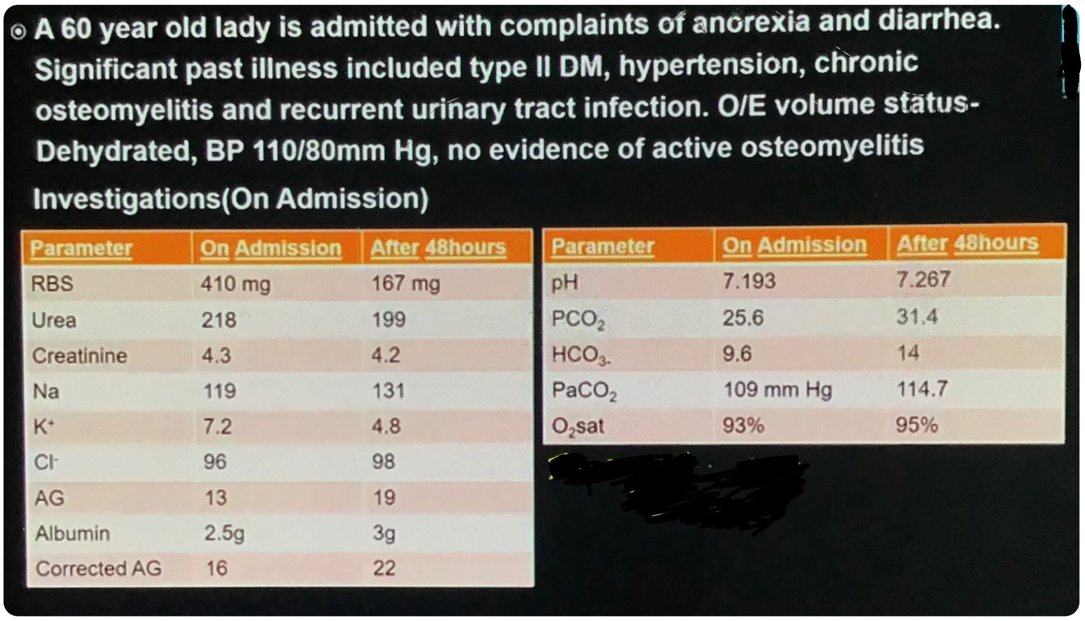
INVESTIGATIONS

WBC : $14500/mm^3$
Hb : 14g
Blood Sugar : 102 mg/dl
Creatinine : 0.8 mg/dl
Na : 129 mmol/L
K : 4.4 mmol/L
Cl : 84 mmol/L
HCO_3 : 15 mmol/L
Case histories : 257

ABG ANALYSIS

pH : 7.21
pCO_2 : 30 mmHg
pO_2 : 85% on room air
HCO_3^- : 14.6 mmol/L
O_2 sat : 80%

Ans :
- Metabolic acidosis + Resp. component
- HAGMA
- Δ Ratio : 2 : Metabolic alkalosis
  HAGMA



- A 60 year old lady is admitted with complaints of anorexia and diarrhea. Significant past illness included type II DM, hypertension, chronic osteomyelitis and recurrent urinary tract infection. O/E volume status- Dehydrated, BP 110/80mm Hg, no evidence of active osteomyelitis [redacted]

Investigations(On Admission)

Parameter	On Admission	After 48hours
RBS	410 mg	167 mg
Urea	218	199
Creatinine	4.3	4.2
Na	119	131
K^+	7.2	4.8
Cl^-	96	98
AG	13	19
Albumin	2.5g	3g
Corrected AG	16	22

Parameter	On Admission	After 48hours
pH	7.193	7.267
PCO_2	25.6	31.4
HCO_3.	9.6	14
$PaCO_2$	109 mm Hg	114.7
O_2sat	93%	95%

[redacted]

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